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THE INSENSIBLE LOSS IN SURGICAL PATIENTS

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SINCE the incidence of reactions to parenteral fluids has been reduced, their value in the treatment of surgical patients has rapidly increased. Although parenteral fluids are widely used today, their administration still remains largely empirical. In order to determine the exact fluid requirements of surgical patients, Coller and Maddock [1, 2, 3] proposed the clinical use of a water balance method described by Soderstrom and DuBois,[4] and by Newburgh, Johnston and Falcon-Lesses.[5] This, essentially, is as follows:

INCREMENTS OF WATER EXCHANGE

Available Water	Excreted Water
(1) Exogenous	(1) Perceptible
(*a*) Diet	(*a*) Urine
(*b*) Water as such	(*b*) Feces
	(*c*) Emesis
(2) Endogenous	(*d*) Drainage
(*a*) Oxidation	(*e*) Perspiration
(*b*) Preformed	
	(2) Insensible

Coller and Maddock[3] have discussed in detail the water balance method. They emphasized especially the importance of the insensible water loss and the necessity of its replacement in the postoperative period. It is this loss which is usually overlooked clinically and which is given further study here.

Benedict and Root[6] define insensible loss as: "The gaseous emanations from the body which do not appear in the form of a sensible moisture or sweat; in other words, the insensible, invisible, intangible but weighable gaseous and vapor productions arising from the lungs in the process of exhalation and from the skin by due process of vaporization." These emanations are chiefly carbon dioxide and water vapor.

Johnston and Newburgh[7] have shown that the insensible weight loss is the resultant of the weight of water lost by vaporization, the weight of the exhaled carbon dioxide, and the weight of the absorbed oxygen; expressed by the following equations:

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$$\text{INSENSIBLE WEIGHT LOSS} = \text{WATER VAPOR} + CO_2 - O_2$$

or

$$\text{INSENSIBLE WEIGHT LOSS} - CO_2 + O_2 = \text{WATER VAPOR}$$

The greater proportion of the insensible weight loss is composed of water. According to Benedict and Root[6] it is about 85 per cent, and to Newburgh, *et al.*,[8] the water vapor varies from 85 to 100 per cent of the insensible loss, depending upon the amount of carbohydrate, protein and fat being metabolized.

Newburgh and his associates[8, 9] have determined that 25 per cent of the total body heat was removed by the vaporization of water. In another study[10] they could demonstrate no reduction in the amount of water vaporized when dehydration of as much as 6 per cent of the total body weight had been produced. It appears, therefore, that the weight of the vaporized water is proportional to the total energy exchange of the body and is not influenced by lesser degrees of dehydration.

Recorded determinations[3] of insensible loss in surgical patients vary from 1,000 to 2,500 Gm. daily, whereas the recorded average[8] losses of normal adults at normal activity vary between 1,000 and 1,500 Gm. daily. It would seem that the total energy exchange of normal active adults would be greater than surgical patients confined to bed, and in view of the above work the insensible loss of the active adults should be greater. Because of this apparent contradiction and the clinical importance of this form of fluid loss, it was felt that further study of the insensible loss in surgical patients was indicated.

The Method and the Control.—In a study of sick surgical patients the method must be adapted to the welfare of the patient and at the same time record the effect of changing intrinsic and extrinsic conditions. An indirect method described by Wiley and Newburgh[11] was, therefore, used in this study. The method is summarized by the following mathematical equations:

$$\text{INITIAL WEIGHT} + \text{TOTAL INTAKE}$$

minus

$$\text{PERCEPTIBLE OUTPUT} + \text{FINAL WEIGHT}$$

equals

$$\text{INSENSIBLE LOSS}$$

The patients were weighed daily at the same hour on a silk balance accurate to $\pm$ 5 Gm. All intake and output was weighed and careful watch was maintained so that nothing was lost before weighing. Weights were measured and checked by more than one observer before recording. Insensible loss and sweating are two distinct and independent processes,[6, 12, 13] hence perspiration was carefully separated from the insensible loss. Bedding and clothing were weighed dry and as soon as perceptible moisture appeared, they were weighed again to determine the loss due to sweat. An effort was made to avoid sweating* by discarding all excess bedding. In the immediate postoperative period,

* Newburgh, *et al.*,[8] have shown that the temperature and the humidity of the environment had little effect on the insensible loss when the subject was clothed and comfortable. All studies were made during the winter and early spring months.

when sweating is apt to occur, the patients were protected from drafts and kept comfortably covered.

Since such factors as body temperature, activity in bed and caloric exchange could not be controlled in the sick surgical patient, determinations were made over several 24-hour periods. The more prolonged observations reflect the result of changing conditions, and in combination give a record of insensible loss under average surgical conditions. Although the insensible loss varies from day to day,[14] the losses of any one case on various days will be found to

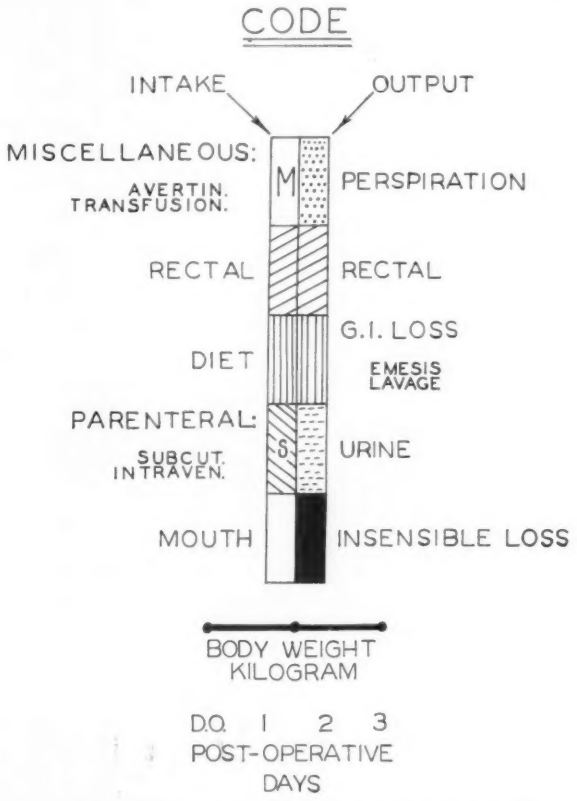


CHART 1.—The Code: Each column represents a 24-hour period. The left hand portion is divided into the increments of intake, the right hand portion into the increments of output. The solid black line represents the cumulative effect of daily fluctuations in body weight.

fall within a small range of fluctuation. Rather wide variations occur on only a few days and these will be discussed separately.

As previously pointed out, the percentage of water vapor in the total insensible weight loss varies with the carbohydrate, protein and fat being oxidized. The amount and proportion of these substances oxidized varies from day to day. Under surgical conditions the mixture oxidized and the weight of the CO_2 and O_2 could not be ascertained. Hence all losses recorded are losses of insensible weight. From a clinical standpoint, the insensible weight loss

may be considered as water loss, as the difference between the two, due to the CO_2 and O_2 exchange, is relatively small.

Observations and Data.—All determinations were graphed on the same scale, the code (Chart 1) describing the various increments.

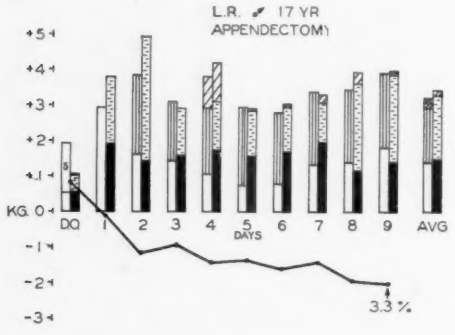


CHART 2.—Case L. R. (P. H. No. 269427): Acute appendicitis.

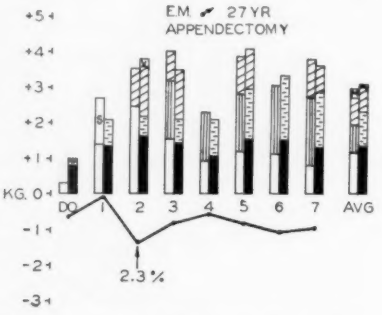


CHART 3.—Case E. M. (P. H. No. 510618): Acute appendicitis.

Case L. R.—(P.H. No. 269427) Acute Appendicitis: There was a rapid post operative recovery. The maximum temperature* of 100.8° F. occurred on the second day. The weight loss continued to 3.3 per cent of the original body weight in spite of a good caloric intake the last seven days. The average insensible loss was 1,503 Gm. per day. The loss on the day of operation does not represent a full 24 hour period (Chart 2).

Case E. M.—(P.H. No. 510618) Acute Appendicitis: The postoperative course was uncomplicated. The temperature curve reached a maximum of 101.8° F. on the first day. The average daily insensible loss was 1,341 Gm. (Chart 3).

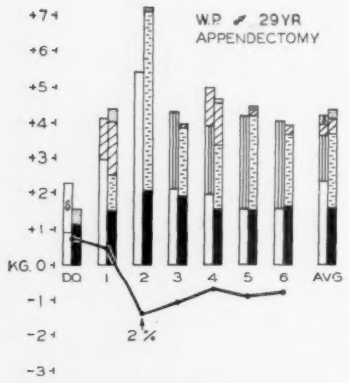


CHART 4.—Case W. P. (P. H. No. 318340): Recurrent appendicitis.

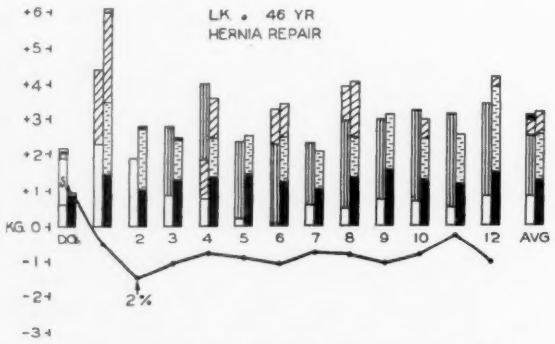


CHART 5.—Case L. K. (P. H. No. 509519): Left indirect inguinal hernia.

Case W. P.—(P.H. No. 318340) Recurrent Appendicitis: The patient had a smooth postoperative course with moderate perspiration the first two days but slight thereafter. The cumulative body weight loss reached a maximum of 2 per cent on the second day coincident with a large fluid intake by mouth and a diuresis of 5,090 Gm. The maximum insensible loss of 2,034 Gm. occurred on the same day. The average daily insensible loss was 1,638 Gm. (Chart 4).

* All temperatures recorded were obtained per rectum.

Case L. K.—(P.H. No. 509519) Left Indirect Inguinal Hernia: The postoperative course was uncomplicated. The maximum temperature, of 101° F., occurred on the first day. The insensible loss averaged 1,256 Gm. per day (Chart 5).

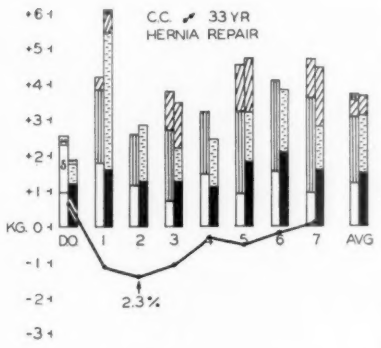


CHART 6.—Case C. C. (P. H. No. 505551): Bilateral inguinal hernia; duodenal ulcer; left herniorrhaphy.

Case C. C.—(P.H. No. 505551) Bilateral Inguinal Hernia, Duodenal Ulcer, Left Herniorrhaphy: There was a minimal postoperative reaction with the maximum temperature, of 100.8° F., occurring on the first day. Maximum body weight loss of 2.3 per cent followed a diuresis on the first day of 3,860 Gm. The insensible loss which averaged 1,498 Gm. per day was somewhat higher the last three days when the caloric intake averaged over 2,900 calories per day (Chart 6).

Case C. C.—(P.H. No. 505551) Right Herniorrhaphy: second operation (ref. Chart 6): There was practically no postoperative reaction. A diuresis of 3,400 Gm. on the first day was followed by the maximum body weight loss, of 2.5 per cent. Again the insensible loss was slightly higher on the last four days, during a period of high caloric intake and increased activity in bed. The average daily insensible loss was 1,665 Gm. The average for both operations, covering a period of 22 days, was 1,604 Gm. (Chart 7).

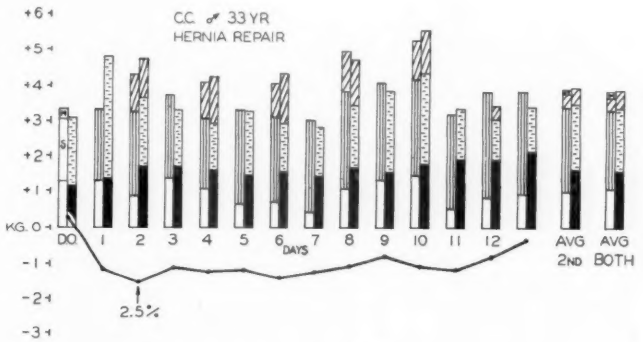


CHART 7.—Case C. C. (P. H. No. 505551): Second operation (ref. Chart 6). Right herniorrhaphy.

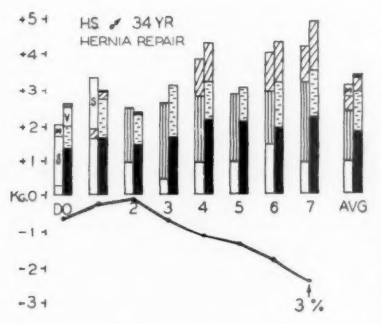


CHART 8.—Case H. S. (P. H. No. 507115): Bilateral inguinal hernia; right herniorrhaphy.

Case H. S.—(P.H. No. 507115) Bilateral Inguinal Hernia, Right Herniorrhaphy: There was a mild postoperative reaction. The maximum temperature, of 101.8° F., occurred on the first day. There was progressive loss of 3 per cent of the original body weight. The average daily insensible loss for the period was 1,804 Gm. (Chart 8).

Case H. S.—(P.H. No. 507115) Left Herniorrhaphy: second operation (ref. Chart 8): There was a minimal postoperative reaction with the maximum temperature, of 99.8° F., occurring on the first day. There was a slow progressive loss of 4.6 per cent of the original body weight even though the caloric intake averaged 2,785 calories

for the last eight days. The average daily insensible loss for the second operation was 1,845 Gm. and for both operations, a period of 22 days, 1,830 Gm. (Chart 9).

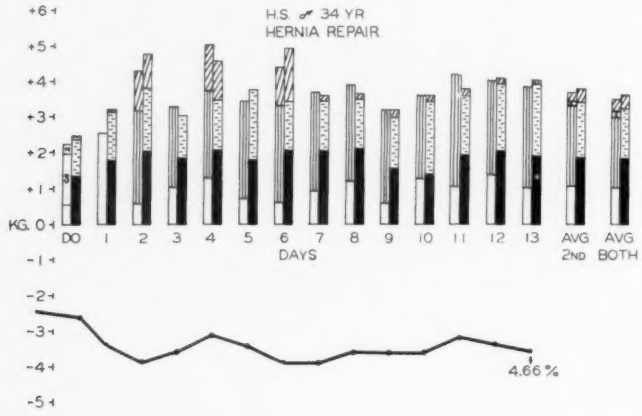


CHART 9.—Case H. S. (P. H. No. 507115): Second operation (ref. Chart 8). Left herniorrhaphy.

Case F. M.—(P.H. No. 493334) Cholecystectomy, Appendicectomy: There was a moderate postoperative reaction the first three days with slight perspiration on the first day. The maximum temperature was 102.8° F. There was a gain of 1,730 Gm. in weight the first two days. This was probably due to water retention as the urinary output was low. On the third day the urinary output increased, the insensible loss was high and the weight started to decrease to a maximum loss of 3.6 per cent. The insensible loss was high on the third and thirteenth days. The average daily insensible loss for the 15 days was 1,500 Gm. (Chart 10).

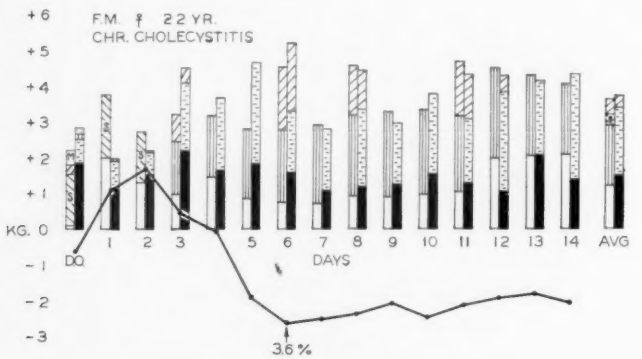


CHART 10.—Case F. M. (P. H. No. 493334): Cholecystectomy; appendicectomy.

Case E. B.—(P.H. No. 517132) Partial Thyroidectomy: This patient presented clinical signs and symptoms of hyperthyroidism with an elevated basal metabolic rate. There was clinical improvement during the preoperative period associated with a small gain in weight. On the day of operation there was a gain in weight of 1,780 Gm. This was probably due to retention for as the urinary output increased on the following day to 3,060 Gm. the body weight decreased 1,920 Gm. The large intake and output on the first day was due to a cool colonic lavage given for a rising temperature, the maximum of which was 102.4° F. The further course was uneventful and the basal metabolic rate

on the sixth day was plus four. The daily average insensible loss for the preoperative period was 1,467 Gm., for the postoperative period 1,321 Gm., and for the 15 day period the average was 1,399 Gm. (Chart 11).

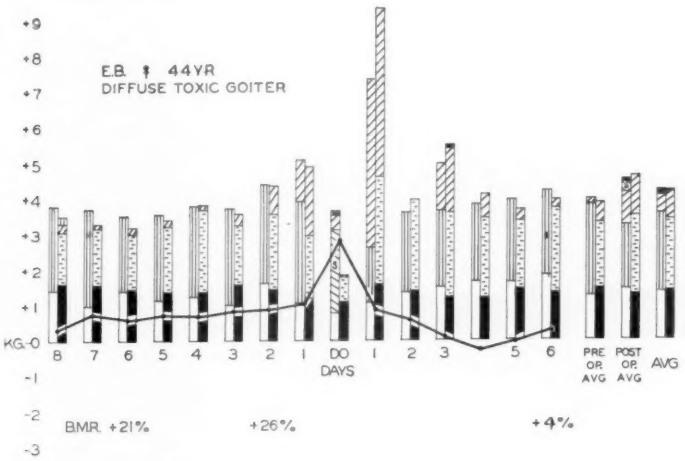


CHART 11.—Case E. B. (P. H. 517132): Partial thyroidectomy.

Case I. E.—(P.H. No. 512999) Partial Thyroidectomy: This was the postoperative study of an uncomplicated thyroidectomy performed for a diffuse toxic goiter. The basal metabolic rate three days before operation was plus 36. The maximum temperature was 100.4° F. on the first day. The basal metabolic rate on the seventh postoperative day was plus three. The daily average insensible loss was 1,154 Gm. (Chart 12).

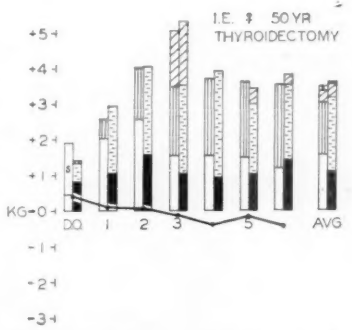


CHART 12.—Case I. E. (P. H. No. 512999): Partial thyroidectomy.

Case E.H.—(P.H. No. 302641) Partial Thyroidectomy: This case presented typical signs and symptoms of hyperthyroidism. The initial basal metabolic rate was plus 47, and the insensible loss 2,258 Gm. These decreased before operation to plus 27 and 1,610 Gm., respectively. There was a slight postoperative reaction, the maximum temperature reaching 101.8° F. on the second day. On the fifth day the patient became emotionally upset. The insensible loss on this day was increased and the basal metabolic rate at the end of the 24 hour period was plus 16. The preoperative daily insensible loss averaged 1,889 Gm. The postoperative daily insensible loss averaged 1,244 Gm. The average daily loss for the 16 days was 1,647 Gm. (Chart 13).

Case P. P.—(P.H. No. 503254) Exploratory Celiotomy, Biopsy of Pylorus, Anterior Gastrojejunostomy: There was a moderate reaction during the first few postoperative days with an uneventful but slow convalescence. The maximum temperature was 101.8° F. on the first postoperative day.

There was a progressive gain of 13.1 per cent of the body weight up to the fourth postoperative day. This was associated with a low urinary output, gastric retention and peripheral edema. At the same time the patient was thirsty and the tongue and skin were dry. There was evidence of hemoconcentration as shown by the increasing hematocrit, plasma specific gravity and plasma proteins determinations.

Parenteral fluids for six days consisted of physiologic saline and Ringer's solutions. There was definite salt retention until the fourth postoperative day, even though

the urinary chlorides, as determined by the Mohr[15] test, as discussed by Fantus,[16] increased to 2.4 per cent. On the fifth day, following administration of 5 per cent dextrose solution, improvement took place as evidenced by loss in weight, increase in the urinary output, disappearance of edema and reduction in hematocrit and specific gravity determinations. Furthermore, all clinical evidences of dehydration disappeared.

It is felt that adequate fluids were administered but the patient received an excessive amount of sodium ions, consequently fluid was retained in the tissues.[17, 18, 19, 20] The gastric retention may also be attributed to edema of the gastrojejunostomy stoma and intes-

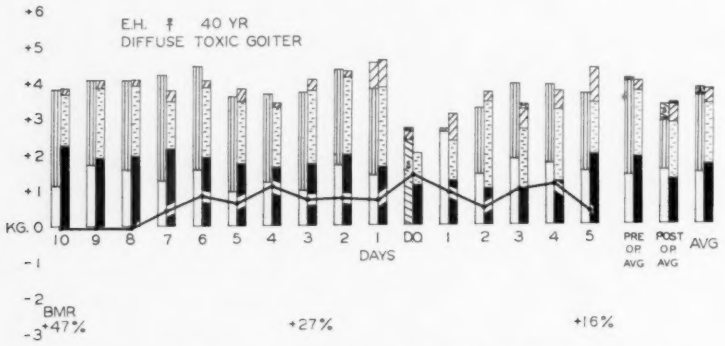


CHART 13.—Case E. H. (P. H. No. 302641): Partial thyroidectomy.

tinal wall as described by Mecray, Barden and Ravdin.[21] Coller and Maddock,[22] and Nadler[23] have discussed in detail the relative value of fluids in surgical patients.

The insensible loss averaged 1,180 Gm. per day for the 21 day period (Chart 14).

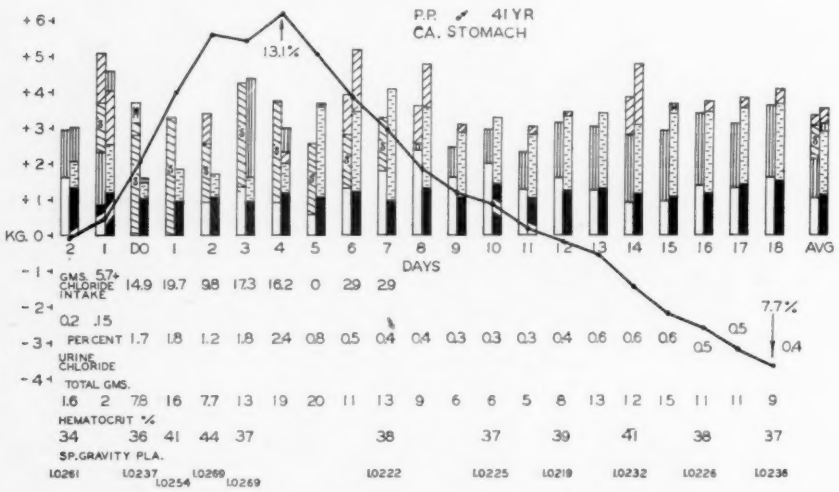


CHART 14.—Case P. P. (P. H. No. 503254): Exploratory celiotomy; biopsy of pylorus; anterior gastrojejunostomy.

Case J. T.—(P.H. No. 509921) Taking Down of Gastro-Enterostomy, Gastrorrhaphy, Partial Jejunectomy, End-to-End Jejunojejunostomy: There was a mild postoperative reaction. The maximum temperature, of 102.8° F., with but one exception occurred on the first day. The insensible loss was low on the day of operation and continued throughout the first three postoperative days during which time clinical evidences of dehydration

were present. Manchester, *et al.*,[24] and Levine and Wyatt[25] have demonstrated the effect of severe dehydration in reduction of the insensible loss. Newburgh and Johnston[10] could demonstrate no effect on the water of vaporization by dehydration of as much as 6 per cent. Hence it must be presumed that prior to this four day period the patient had lost more than 6 per cent of his body weight in fluids.

On the second day, when the loss was lowest, the patient presented a typical picture of severe dehydration and passed 110 Gm. of urine during the first 12 hours. The pulse suddenly increased to 150 and the temperature to 104° F. After an infusion there was definite clinical improvement, the pulse and temperature fell and the urinary output, for the remaining 12 hour period, was 1,020 Gm.

The patient maintained his weight to the sixth postoperative day, at which time parenteral fluids containing salt were stopped and a diuresis developed. Weight loss continued until the twelfth day, when it was 7.9 per cent of the body weight. Sodium chloride capsules were administered by mouth and the weight slowly increased, but it was

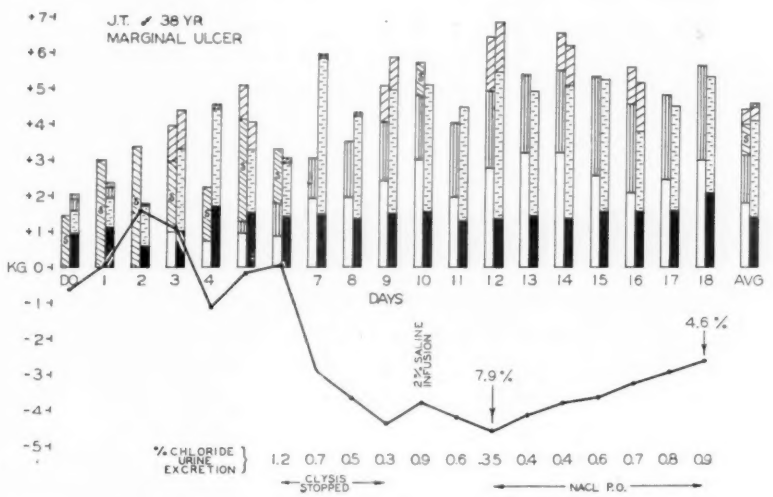


CHART 15.—Case J. T. (P. H. No. 509921): Taking down of gastro-enterostomy; gastrorrhaphy; partial jejunectomy; end-to-end jejunojejunostomy.

not until the sixteenth postoperative day that the diuresis stopped and the chloride excretion rose above normal.

It is believed that the conditions in this case are the converse of the previous case; namely, salt starvation. It might be added that for 37 days prior to operation the patient received medical treatment for the marginal ulcer during which time the nature of the diet included but little, if any, sodium chloride.

The average daily insensible loss for the 19 day period was 1,402 Gm. (Chart 15).

The average insensible loss per day in the series ranged from 1,154 Gm. to 1,830 Gm. The average for 175 twenty-four hour periods was 1,457 Gm. Of the total output of each case the insensible loss ranged from 30.8 per cent to 49.6 per cent or an average for all cases of 39.4 per cent (Table I).

Benedict and Root[6] have shown that the insensible loss is approximately proportional to the body weight. Levine, *et al.*,[26] have also shown a similar relationship between insensible loss and body weight, height and surface area. The cases considered in this study were arranged in order of their body weight irrespective of the type of the surgical procedure. Although the study was

not conducted with the patients at basal conditions, there exists an apparent relationship between the size of the body and the insensible loss.

There were two cases which did not follow this relationship. The first, Case F. M. (Chart 10) was a female, age 22, five feet three inches tall, weighing 161 pounds. She had chronic cholecystitis and cholelithiasis. Her stature and the disease suggest a metabolic disturbance. The daily insensible

TABLE I

SUMMARY OF INSENSIBLE LOSS DETERMINATIONS

Case	Height	Weight in Kilograms	Sur. Area Sq. Meter	Days	Average Daily Insensible Loss			Insensible Loss Percentage of Total Output
P. P.	5′ 2 ″	47.57	1.45	21		1,180		33.2
I. E.	5′ ¾″	49.08	1.43	7		1,154		32.4
E. H.	5′ 1¾″	49.24	1.48	10	Preop.	1,889	47.2	
				6	Postop.	1,244	37.0	
					Ave.	1,647		44.0
E. B.	5′ 3⅜″	50.95	1.51	8	Preop.	1,467	38.3	
				7	Postop.	1,321	28.6	
					Ave.	1,399		33.3
E. M.	5′ 6¼″	56.59	1.64	8		1,341		43.6
J. T.	5′ 8¼″	57.29	1.67	19		1,402		30.8
L. R.	5′ 7¾″	61.50	1.73	10		1,503		44.0
C. C.	5′ 3¾″	63.33	1.67	8	1st op.	1,498	40.0	
				14	2nd op.	1,665	42.6	
					Ave.	1,604		42.0
W. P.	5′11 ″	66.47	1.85	7		1,638		38.0
F. M.	5′ 3⅛″	73.44	1.76	15		1,500		40.2
L. K.	5′ 8¼″	75.10	1.88	13		1,286		41.0
H. S.	5′10¾″	78.63	1.98	8	1st op.	1,804	52.0	
				14	2nd op.	1,845	43.5	
					Ave.	1,830		49.6
				175	Days—Average—	1,457 Gm.		39.4

loss curve was the most irregular of the series. For the size of the patient the average insensible loss for the period was low. The second, Case L. K. (Chart 5), although well proportioned, had a thick, flabby panniculus adiposus. He was phlegmatic in type and extremely inactive both mentally and physically. For the size of the patient the average insensible loss for the period was extremely low.

It is interesting to note that the hyperthyroid cases, after surgical correction of the metabolic disturbance, fell into the proper relationship according to the size of the patient. Prior to operation the insensible loss of these cases was abnormally high.

Discussion.—The amount of fluids ordered for a surgical patient is usually determined by the appearance of the patient and the amount of intake and output recorded on the chart for the previous day or days. Respectful attention is always given to the urinary output for the preceding 24 hours before the fluid program is outlined for the day.

The following case illustrates graphically the inadequacy of such a program and the clinical importance of other than perceptible losses.

Case S. L.—(P.H. No. 448691) Enterostomy, Exploratory Celiotomy, Release of Peritoneal Band: The figures which were accepted as a daily guide for fluid administration were taken from the chart and plotted. The difference between the daily intake and output was represented as a cumulative increase or decrease in weight. The cumulative gain of intake over output amounted to 26.7 Kg., or 37 per cent of the total weight of the patient. It is improbable that the patient retained fluids amounting to 59 pounds for there was an obvious decrease in the weight of the patient during his illness (Charts 16 and 17).

In Chart 17 it will be noted that the intake and the output are the same as in Chart 16, except that to each daily output was added an estimated loss of 1,500 Gm. per day. The daily output then exceeded the intake and the curve of the cumulative difference decreased constantly to a point which was 14 per cent below the original weight of the patient. At this point the patient suddenly went into a state of collapse; the extremities became cold, cyanosis was marked, the blood pressure fell, the pulse was rapid and thready and the temperature and respirations increased rapidly. The patient was placed in an oxygen tent and an infusion administered until a transfusion could be given. As a result of this treatment the patient slowly responded and the convalescence thereafter was slow and uneventful. Throughout the remaining postoperative course intake exceeded output, and the weight curve slowly returned toward the normal.

This case demonstrates three important facts; namely, The empiric administration of fluids to be unsatisfactory; the false security of a urinary output which seems to be adequate; and the clinical importance of the insensible loss. It is felt that the collapse of the patient was due to severe dehydration. Eight hours after collapse, and after the above emergency measures were instituted, the specific gravity of the blood was still as high as 1.0618. What the specific gravity was when the collapse occurred is unknown. Coller and Maddock[27] have demonstrated, in humans, that after 6 per cent of the body weight in fluids has been lost the subject is on the verge of serious dehydration. Usually urinary output diminishes with progressive dehydration, as demonstrated by Case P. P. (Chart 14) and Case J. T. (Chart 15). In Case S. L. (Chart 17) the urinary output was more than a liter per day. This was considered adequate and parenteral fluids were stopped on the eighth postoperative day. The reserve supply of fluid which was already diminished continued to fall more rapidly, during the next two days, to a maximum loss of 14 per cent. The body was then so depleted of fluid that severe hemoconcentration and collapse occurred.

As previously pointed out, in this series of cases, 39.4 per cent of the total output was lost through the insensible route. This is sufficient to make the insensible loss as serious a dehydrating factor as the urinary output, as demonstrated in Case S. L. (Charts 16 and 17). Intake exceeded output by far until allowance was made for the insensible loss, at which time a marked

negative balance occurred. It is to be remembered that the dissipation of heat through the vaporization of water continues constantly until the water supply is depleted and a critical state of dehydration exists.

CHART 16

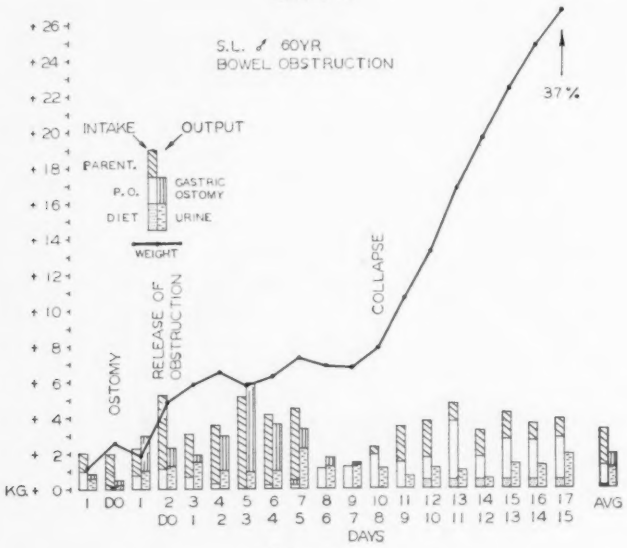
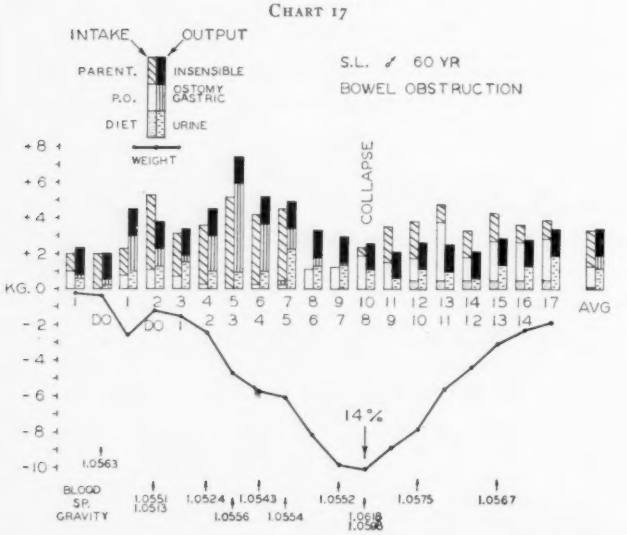


CHART 17



CHARTS 16 and 17.—Case S. L. (P. H. No. 448691): Enterostomy; exploratory celiotomy; release of peritoneal band.

From a surgical point of view the most common form of deranged water exchange is that of dehydration. The nature of some surgical procedures necessitates a method of therapy which contraindicates the administration of fluid by the usual routes. Frequently this deprivation extends over a period of several days and sometimes a week or longer. Occasionally, persistent vomiting, prolonged enterostomy drainage or forced duodenal drainage is

required which also depletes the body of fluid and electrolytes. Unless fluid is adequately administered by other routes, a state of dehydration is inevitable.

The patients undergoing less extensive surgical procedures are usually capable of maintaining a satisfactory balance of water and salt. The cases in which major procedures are undertaken, especially those on the gastrointestinal tract or those which disturb the normal function of the tract, are dependent upon the judgment of the surgeon for fluid, electrolytes and nourishment.

If dehydration is to be avoided rather than combated, adequate provision must be made to cover these losses, including that lost insensibly. It is impractical to determine the insensible loss of each surgical patient but a reasonable amount, as demonstrated in this study, can be estimated from the size and weight of the patient. Only by recognizing the clinical importance of the insensible loss can replacement be made according to the physiologic requirements of the surgical patient.

SUMMARY

(1) The insensible loss in 12 surgical patients having 14 operations ranged from 1,154 to 1,830 Gm. per day.

(2) The average insensible loss for 175 twenty-four hour periods was 1,457 Gm. per period.

(3) The insensible loss for all the cases averaged 39.4 per cent of the total output.

(4) The insensible loss varied with the size of the patient rather than with the type or the extent of the surgical procedure.

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BIBLIOGRAPHY

[1] Coller, F. A., and Maddock, W. G.: Dehydration Attendant on Surgical Operations. J.A.M.A., **99**, 875, 1932.

[2] Coller, F. A., and Maddock, W. G.: The Water Requirements of Surgical Patients. ANNALS OF SURGERY, **98**, 952, 1933.

[3] Coller, F. A., and Maddock, W. G.: Water Balance in Surgical Conditions. Internat. Clin., **3**, 191, 1934.

[4] Soderstrom, G. F., and DuBois, E. F.: The Water Elimination Through Skin and Respiratory Passages in Health and Disease. Arch. Int. Med., **19**, 931, 1917.

[5] Newburgh, L. H., Johnston, M. W., and Falcon-Lesses, M.: Measurement of Total Water Exchange. Jour. Clin. Invest., **8**, 161, 1930.

[6] Benedict, F. G., and Root, H. F.: Insensible Perspiration: Its Relation to Human Physiology and Pathology. Arch. Int. Med., **38**, 1, 1926.

[7] Johnston, M. W., and Newburgh, L. H.: The Determination of the Total Heat Eliminated by the Human Being. Jour. Clin. Invest., **8**, 147, 1930.

[8] Newburgh, L. H., Wiley, F. H., and Lashmet, F. H.: A Method for the Determination of Heat Production Over Long Periods of Time. Jour. Clin. Invest., **10**, 703, 1931.

[9] Newburgh, L. H., Johnston, M. W., Lashmet, F. H., and Sheldon, J. M.: Further Ex-

periences with the Measurement of Heat Production from Insensible Loss of Weight. Jour. Nutrit., **13,** 203, 1937.

[10] Newburgh, L. H., and Johnston, M. W.: Relation Between Dehydration and the Insensible Loss of Water. Jour. Nutrit., **7,** 107, 1934.

[11] Wiley, F. H., and Newburgh, L. H.: An Improved Method for the Determination of Water Balance. Jour. Clin. Invest., **10,** 723, 1931.

[12] Vasti, Assunta: The Insensible Water Loss through the Skin. Am. Jour. Physiol., **102,** 60, 1932.

[13] Hancock, W., Whitehouse, A. G. R., and Haldane, J. S.: The Loss of Water and Salts through the Skin and the Corresponding Physiological Adjustments. Proc. Roy. Soc. London, sB., **105,** 43, 1929.

[14] Lavieties, P. H.: The Metabolic Measurement of Water Exchange. Jour. Clin. Invest., **14,** 57, 1935.

[15] Peters, J. P., and Van Slyke, D. D.: Quantitative Clinical Chemistry. Vol. *2*; Williams and Wilkins Company, Baltimore, 1932.

[16] Fantus, Bernard: Fluids Postoperatively. J.A.M.A., **107,** July 4, 1936.

[17] Matas, Rudolph: The Continued Intravenous Drip. ANNALS OF SURGERY, **79,** 643, 1924.

[18] Keefer, C. S.: Edema: Its Pathologic Physiology and Clinical Significance. Internat. Clin., **2,** 85, 1933.

[19] Davis, H. A., and Dragstedt, L. R.: The Relative Significance of Electrolyte Concentration and Tissue Reaction in Water Metabolism. Am. Jour. Physiol., **113,** 193, 1935.

[20] Curphey, W. C., and Orr, T. G.: Edema in Surgical Patients. Surgery, **1,** 589, 1937.

[21] Mecray, P. M., Barden, R. P., and Ravdin, I. S.: Nutritional Edema: Its Effect on the Gastric Emptying Time before and after Gastric Operations. Surgery, **1,** 53, 1937.

[22] Coller, F. A., Dick, V. S., and Maddock, W. G.: Maintenance of Normal Water Exchange with Intravenous Fluids. J.A.M.A., **107,** 1522, November 7, 1936.

[23] Nadler, S. B.: The Use of Parenteral Fluids. Surgery, **1,** 964, 1937.

[24] Manchester, R. C., Husted, C., and McQuarrie, I.: Influence of the State of Hydration of the Body on the Insensible Loss of Weight in Children. Jour. Nutrit., **4,** 39, 1931.

[25] Levine, S. Z., and Wyatt, T. C.: Insensible Perspiration in Infancy and in Childhood. IV. Basal Measurements in Dehydrated Infants. Am. Jour. Dis. Child., **44,** 732, 1932.

[26] Levine, S. Z., Kelly, M., and Wilson, J. R.: The Insensible Perspiration in Infancy and in Childhood. II. Proposed Basal Standards for Infants. Am. Jour. Dis. Child., **39,** 917, 1930.

[27] Coller, F. A., and Maddock, W. G.: A Study of Dehydration in Humans. Trans. Am. Surg. Assn., **53,** 460, 1935.

EXCISION OF THE AXILLARY VEIN IN THE RADICAL OPERATION FOR CARCINOMA OF THE BREAST*

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THIS communication presents an argument in favor of the excision of the axillary vein during so-called radical breast operations when involved lymph nodes lie in close or immediate proximity to the vein. The question of indications for or contraindications to radical mastectomy for carcinoma will not be considered, the present discussion being concerned solely with the management of the axillary vein when the radical operation is contemplated. It is quite generally agreed that in the effort to remove the fat and the lymph nodes completely during the customary dissection of the axilla, the axillary vein must be more or less bared and stripped clean. The closer the involved nodes lie to the axillary vein, the greater is the necessity of extensively freeing the vein. The latter has been injured occasionally during this dissection and, according to case reports, has been ligated without untoward effect under such circumstances. On the other hand, no reports have been found which dealt with deliberate excision of the axillary vein in order to achieve a more thorough block removal of the axillary contents.

The argument in favor of excision of the axillary vein in selected cases is simple and can be stated briefly: Although extirpation of every cancerous nest by means of a careful axillary dissection is not to be anticipated, an effort is always made to effect a complete removal of the axillary contents. When there is minimal axillary lymph node involvement or when the invaded lymph nodes are not situated near the vein, the standard dissection may meet all requirements. On the other hand, one experiences a sense of inadequacy during such dissections when involved nodes are close to or lie upon the vein. The likelihood or possibility of leaving on the vein small, perhaps microscopic fragments of cancerous tissue is great. Furthermore, cancer cells within or cancerous involvement of lymph channels represent part of the picture of cancerous invasion of the axilla. When the lymphatic network, which has a well-known tendency to encircle the axillary vein, is involved in immediate proximity to the vein (which is very likely to be the case when cancerous lymph nodes are situated close to the vein), removal of such lymphatic tissue by any procedure other than by removal of the axillary vein appears improbable. It may be argued that, under the foregoing circumstances, a radical operation is futile and the addition of a more radical procedure, purposeless. This view may be correct in one case, and false in another, for there is no way at the present time to prognosticate the ultimate outcome of an operation in any

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given case when involved nodes are situated near the axillary vein. Therefore, as long as the possibility of eradication exists, the objective should be the widest possible block removal of visibly invaded tissue. That purpose is not achieved if involved nodes in close proximity to the axillary vein (with or without involved intercommunicating lymphatics) are dissected from the vein, no matter how cleanly the latter may be stripped by a painstaking technic. In order to attempt to achieve complete removal, the objective is more nearly approached by removal of the vein together with the involved nodes and axillary fat in one block. Thus it appears quite as logical to excise the axillary vein in the block removal of cancerous axillary lymph nodes, as to excise, in the generally practiced manner, the internal jugular vein in the block removal of cancerous cervical lymph nodes.

The deterrent factor in a consideration of excision of the axillary vein unquestionably is the fear of interference with the circulation which might ensue. The belief that obstruction of the main venous trunk of an extremity leads to edema is based presumably on the presence of edema in cases of thrombophlebitis in an upper or lower extremity. Little thought appears to have been given to the fact that perivenous lymphangitis usually exists in cases of thrombophlebitis. Furthermore, there can be no doubt that lymphatic blockade produces edema of an extremity. In any event, the idea that the obstruction of a vein inevitably results in edema is incorrect. The writer has shown and reported[1, 2, 3] cases of excision of the axillary, femoral, and iliac veins (for suppurative thrombophlebitis), and has demonstrated that edema of the upper or lower limb respectively did not exist before operation and did not occur after operation.

Despite a logical basis for excision of the axillary vein, it was a far cry from a patient desperately ill from a suppurative phlebitis and septicemia, to one with carcinoma of the breast who might not only not be helped but possibly be harmed by such a procedure. For this reason removal of the axillary vein as part of block removal of the axillary contents was performed in the beginning solely in instances of extensive axillary node involvement. When excision of the vein proved harmless in these earlier cases, the procedure was extended to cases of less extensive perivenous lymph node involvement and to cases in which the nodes did not lie in immediate proximity to the vein.

A brief description of the technic of excision of the axillary vein in the block removal of the axillary contents is sufficient, as no unusual operative procedures have been employed. Nothing is added to the risks of the radical operation; indeed, the dissection of the axilla is aided and simplified. As soon as the axilla is entered, the relationship of involved nodes (if any) to the axillary vein is ascertained. If excision of the axillary vein is decided upon, it is exposed at the apex of the axilla and severed between ligatures; it is again severed in its third portion, in the same manner, at the lateral limit of the axilla. The usual dissection of the axillary contents, after severance of the axillary vein, is simplified, naturally, by the elimination of the dissection required for the removal of nodes about the vein. As a result, the duration

of operation is materially shortened. The existence of a double axillary vein may be recognized only after the more superficial vessel has been divided and freed.

Special histologic studies of the removed vein and adjacent tissues have not been carried out. They might have proved conclusively the advantages which I have claimed for the procedure in question. Of course, the pathologic material available from the small number of cases in which excision of the vein has been practiced might also have failed to supply conclusive evidence. There was, however, a significant finding in one specimen removed at operation. Immediately adjacent to the vein was a lymph node which was so minute that it could be seen macroscopically, as a speck, only in the stained section. It is interesting to note that microscopically part of this node was found to be invaded by cancer and part was uninvolved. Whether this node, not visible in the operative field or in the gross specimen, would or would not have been removed by the standard dissection about the axillary vein cannot be stated definitely. One may assume that there was an equal chance that it would have been left behind, particularly because of its intimate relationship to the axillary vein.

Concerning the question of improvement in end-results by the addition of excision of the axillary vein to the customary radical operation, any statement made at this time would be entirely valueless, because the procedure has been employed only in 11 cases. A definite statement can, however, be made concerning the question of edema following operation. Some edema of the upper arm followed the operation in two instances, and there was fluctuating edema of the hand in two other cases. This represents, perhaps, the incidence of edema following the radical operation when the axillary vein is not excised. However, exact figures are of no moment for we are not concerned with statistics. The essential innocuousness of removal of the axillary vein is established even by the small number of cases in which it has been undertaken. As stated at the outset the purpose of this communication was to set forth an argument in favor of excision of the axillary vein when cancerous lymph nodes lie in close or immediate proximity to the vein. The argument appears logical and to that extent warrants the belief that improvement in the end-results might come to pass if the proposed step were added, in selected cases, to the standard dissection of the axilla in the radical operation for cancer of the breast.

REFERENCES

[1] Neuhof, H.: The Diagnosis and Operative Control of Acute Pyogenic Phlebitis Complicated by General Septic Invasion. ANNALS OF SURGERY, June, 1933.

[2] A Case of Excision of the Common, External, and Internal Iliac Veins for Suppurative Thrombophlebitis, Without the Development of Postoperative Edema. Jour. Mount Sinai Hosp., March–April, 1935.

[3] Excision of Vein for Suppurative Thrombophlebitis. Case Report. ANNALS OF SURGERY, **106**, No. 2, August, 1937.

Discussion.—Dr. Emil Goetsch (Brooklyn, N. Y.), apropos of the question of the edema of the arm, following radical resection of the breast, recalled Doctor Halsted speaking of the causes which he thought influenced this condition. He paid particular attention to the wound healing following radical resection and found, after a study of a considerable series of cases, that in instances in which the wound healed without discharge of serum or seropurulent secretion, indicating a mild infection, edema of the arm did not follow, even in instances in which the axillary vein was involved or possibly ligated. In those cases in which there was even the mildest infection, indicated by the discharge of a cloudy serum, edema of the arm was likely to follow. He came to the definite conclusion that the edema was not due to any particular procedure involving the axillary vein but rather to the occlusion by fibrosis of the lymphatics remaining after the resection. This fibrosis is primarily the result of infection and because of it the possibility of subsequent improvement in the edema is remote. Accordingly, Doctor Halsted felt that the condition of the axillary vein is not the determining factor in the case with edema.

Dr. John E. Jennings (Brooklyn, N. Y.) reported his experience with regard to the procedure under discussion, stating that he had accidentally ligated the vein, probably half a dozen times, with resection of the vein in small segments, as part of the attempt to remove all the axillary contents. Only recently, however, had he with "malice," as Doctor Neuhof described it, removed the vein in very much the way that has been described. Doctor Jennings has done this in six cases within the last three years, two of whom died promptly within three or four days after operation. In one an autopsy was obtained and a clot was found in the subclavian vein, with the rest of the clot, or a considerable portion of it, in the pulmonary artery. In the other case, which was considerably more difficult to do, death occurred in about the same way but no autopsy was obtained. Doctor Jennings felt there is a limited field for this procedure. The exposure of the axilla is better, and its apex can be better cleared of the fascia. There are certain cases where it is almost necessary to do this if one is to accomplish a complete removal of the lymphatics of the axilla after intensive radiation. If one is going to impose radical surgery upon radical radiation one will often find it exceedingly difficult to clear the vein; a complete évidement is only possible by its resection. Eventually the limits of surgery in dealing with cancer of the breast will be found; but a word of caution is not amiss: it is possible to go too far.

Dr. George H. Semken (New York): The studies of Florence Sabin, Huntington and McClure, and von Schulte have shown the close genetic relationship of the lymphatics and the veins. Sabin showed that the jugular lymph sacs, for example, were formed by the coalescence of small veins at the venous confluence in the neck, which were first emptied of their contained blood, and later, after coalescence, again joined the subclavian or internal jugular veins or both. From these sacs, a centrifugal development of lymph vessels then occurred. Eventually, the sacs became groups of lymph nodes. The other observers demonstrated the existence of primordia in various areas of the mesenchyme, from which angioblasts were developed, which became blood vessels and lymph vessels respectively, and developed centripetally. It is almost axiomatic that the lymphatic trunk vessels tend to accompany the veins of the respective regions.

In the relationship of the lymphatics of the breast to the axillary vein—the axillary vein is not properly comparable to the internal jugular vein. In

the neck (excluding the superficial chain of lymphlatics), the submental and submaxillary lymph nodes, which form the first line of defense for their respective drainage areas, are alone distant from the internal jugular vein. The carotid packet of the deep chain, next to these in the lymph stream, is, however, immediately regional to the tongue, fauces and pharynx, epipharynx and the larynx. These nodes lie directly at and upon the internal jugular vein; and from this point downward, the successive nodes in this lymph stream are closely related to the vein. Many of these nodes have efferents directly into the vein. Early fixation of invaded lymph nodes to the vein is, therefore, frequently found in the neck. In the axilla, however, the relations of the mammary lymphatic vessels and node groups to the axillary vein are different. The lymph nodes immediately regional to the breast are the anterior thoracic (or pectoral) group. In these nodes, cancer may remain localized for some time. Next in line are the nodes of the central group. From these, the further course is an arc to the axillary vein and then paralleling it to the infraclavicular node group at the clavicle, to end in the subclavian trunks. Fixation of the nodes to the axillary vein is, therefore, relatively long delayed. This anatomic relationship has been clearly demonstrated by Oelsner, Mornard, Poirier, and Most, who injected the breasts of cadavers, mainly of newly born infants. The summarized findings are well shown in the chart of Poirier (reproduced in Gray's Anatomy).

In the usual type-case of mammary cancer that is considered operable, even in the presence of fairly advanced primary tumors and palpable axillary lymph-nodes, it has not been a frequent experience to find cancerous lymph nodes fixed to the axillary vein. There does not seem to be any compelling reason, therefore, to resect the axillary vein routinely in every case. Rather, this resection, partial and not complete, should be reserved for the cases in which there is actual or imminent fixation of the lymph nodes.

The excision of the vein has disadvantages and danger. The obstruction to the venous return is not sufficient to cause much edema, but it delays the early restoration of function in the arm. The danger lies in the possibility of a fatal pulmonary embolism. Embolism is favored by the formation of a large clot in a long, dead pocket in the vein, and a resultant ascending thrombosis; and this is the probable status when the vein segment is excised at the first site of lymph node fixation, in the area of the thoraco-acromial vessels, because of the ligation and section of the upper axillary tributaries in the radical breast operation. This danger is lessened if the upper ligature is placed near the entrance of the small tributary veins at the clavicle; which will leave no noteworthy pocket in which the blood can become stagnant and be clotted. It is desirable, also, to avoid an undue inflammatory reaction about the ligated vein. The vein is, therefore, skeletonized, to minimize the amount of resultant dead tissue in the ligated stump; and a double strand of No. 000 chromic catgut is employed for the ligation, instead of a heavy or nonabsorbable ligature.

When the fixation of cancerous lymph nodes to the axillary vein has necessitated the excision of the related segment of the vein, the microscopic examination of this issue has usually shown that the fixation has been limited to the sheath, without invasion of the vein wall; and this has been the characteristic finding, also, in the secondary operations upon cases recurrent after previously incomplete operations. Evidently then, it is the vein sheath and not the vein that is the site of potential cancer involvement; and this finding seems to make it imperative to remove the accessible parts of the sheath of the axillary and basilic veins with the mobilized tissues, routinely in all pri-

mary, radical operations. This dissection will include a large part of the deep layer of the sheath, where small lymph nodes may lie.

The primary need in the radical breast operation is the complete understanding of the paths of cancer extension, a comprehensive and well ordered plan of operation, and a painstaking anatomic dissection. This should emphasize the wide removal of the kin of the breast, especially in the neighborhood of the tumor; an incision adequate to afford full exposure of each part of the proposed field of operation; thin flaps; the complete removal of the pectoralis major and minor muscles; the special care to clear the anterior intercostal spaces; the precise and methodical clearing of the axillary and subscapular spaces; and the complete removal of the areolar tissue over the floor. Particularly important is the need of care during the operation, to avoid any pressure upon the tumor and the related tissues, which might loosen cancer plugs and give rise to distant metastases.

POSTOPERATIVE ROENTGENOTHERAPY IN CANCER OF THE BREAST*

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IN 1922, the writer,[1] in association with Doctor Peck, and again, personally,[2] in 1927, published articles upon the results of operation on cancer of the breast in which the surgical procedures employed differed from the classic operations of Halsted[3] and Meyer,[4] in that the pectoralis minor was not removed, the amount of excised skin was less, and skin grafting rarely employed. Toward the latter part of the second period[2] subcutaneous dissection, as advocated by Sampson Handley,[5] was more frequently employed. Although our percentage of five-year postoperative freedom from recurrence was comparable to that reported from other clinics, we were subjected to unfavorable criticism on the ground that a proper dissection of the axilla could not be made if the pectoralis minor was allowed to remain in place, and secondly, that our reported local recurrence of 36 per cent was due to the fact that not enough skin had been excised, notwithstanding the employment of the Handley modification. The first criticism was considered valid and subsequently the pectoralis minor was removed with the other tissues en masse, in many instances. It will be noted later that the former procedure has still been employed in many of the cases. It has been felt, however, that with the Handley modification, sufficient skin surrounding the tumor could be removed and still permit closure in all cases except those with small breasts or large tumors.

Surgeons are accustomed to judge the adequacy of their operative procedure by the cure of the disease locally. A recurrence in the operative field has usually been considered a reflection upon the operator. Lewis and Rienhoff,[6] in March, 1932, reported a study of the results of the radical Halsted operation with immediate Thiersch skin graft. In Table XXIX they give complete data on the first site of recurrence in 225 cases. Of these, 79, or 35 per cent, developed a local recurrence. When the technic was varied, only to the extent of removing less skin to allow plastic closure of the wound, there was a local recurrence in 36 out of 77 cases, or 46.7 per cent.

In this series, we have complete data on 119 patients followed for five years, relative to the first site of recurrence. Of this group, 46, or 38 per cent, had local recurrence. We noted, as of special interest, that the recurrence in the axillary region was confined to the cases in which the pectoralis minor had been left in place. Adverse criticism, in a measure, is

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TABLE I

ANALYSIS OF POSTOPERATIVE RESULTS IN 227 CASES OF CARCINOMA OF THE BREAST

January 1, 1922—December 31, 1931

Radical operations	227
Operative deaths	5
Dead intercurrent disease	7
Dead of cancer within five years	123
Alive over five years with recurrence	8
Alive over five years and well	55
Lost (not followed five years)*	29

* Many of the cases lost were favorable cases without metastases, and some had known four-year freedom from recurrence. So that in arriving at a percentage of five-year "cures," the cases lost, and those who died of intercurrent disease have been eliminated. We would then have 55 out of 191 cases, or 27 per cent, alive and well at the end of five years.

probably justifiable if a local recurrence appears. Observation, however, on many cases has convinced the writer that the recurrences in the intercostal spaces near the sternum are the result of retrograde growth from internal mammary nodes that were already involved at the time of the operation. It is also thought that even with the most meticulous and thorough surgery, the incidence of local recurrence without roentgenotherapy will remain around 30 per cent. It has been observed that the cancer is rarely confined to the area of the operative field in instances in which a local recurrence occurs. Indeed, it is not uncommon to find coincident regional or distant metastases.

TABLE II

SITES OF FIRST RECURRENCE IN 119 INSTANCES

Local	46
Other breast	7
Supraclavicular	6
Chest	27
Abdomen	14
Osseous	19
	119

Since 1921, the use of high voltage therapy in the treatment of carcinoma of the breast has received increasing consideration. There are some roentgenologists who are satisfied to rely on roentgenotherapy alone. At the other extreme, there are surgeons who feel that there is no benefit to be derived from roentgenotherapy. The Halsted type of operation had become well established before roentgenotherapy came into vogue, so that we may use the reports from such operations as criteria. Nevertheless, one must bear in mind the variation in the course and character of different breast cancers. Many with no treatment live for years; many with inadequate operation in our group have lived long with no recurrence. On the other

hand, many cases that looked favorable have died within a few months. In this type no form of treatment seemed to be of value.

TABLE III

ANALYSIS OF FIVE-YEAR RESULTS IN THREE CLINICAL TYPES OF 186 CARCINOMATA OF THE BREAST

(A) No Metastases (Axillary)

Roentgen Ray		No Roentgen Ray			
R.*	P.M.†	R.	P.M.		
8	7	1	7	Dead	23
13	15	3	10	O.K.	41
5	0	0	1	Recurrence active	6
26	22	4	18		70

(B) Metastases Not Palpable (Axillary)

Roentgen Ray		No Roentgen Ray			
R.	P.M.	R.	P.M.		
12	26	3	5	Dead	46
4	4	1	1	O.K.	10
1	0	0	1	Recurrence active	2
17	30	4	7		58

(C) Metastases Palpable (Axillary)

Roentgen Ray		No Roentgen Ray			
R.	P.M.	R.	P.M.		
16	20	4	14	Dead	54
2	2	0	0	O.K.	4
0	0	0	0	Recurrence active	0
18	22	4	14		58

Total roentgen-rayed—No metastases	48	= 28 O.K. = 58%
No roentgen-ray—No metastases	22	= 13 O.K. = 59%
Total roentgen-rayed—Metastases	87	= 12 O.K. = 13%
No roentgen-ray—Metastases	29	= 2 O.K. = 3%
	186	

* R. = Radical operation.

† P.M. = Pectoralis minor allowed to remain with removal of less skin.

Roentgenotherapy alone has been used, so far, on such small groups of cases that no definite final opinion may be expressed.

Interstitial radium therapy alone has been employed for some years by Keynes.[7, 13] Recently he wrote:[13] "I wish to emphasize again the fact that interstitial radium treatment is strictly comparable with surgical operation, in that it is a local form of treatment. For this reason no striking improvement in the survival rate was to be expected if radium was to be used as an alternative to surgery. It is the metastases, and not the primary disease, that usually cause the death of the patient, and for that reason I never shared the exaggerated hopes that were one time placed by some people in the future

of radium." He has published[13] a five-year report on 201 cases. When the disease was confined to the breast, clinically, 71.4 per cent (of 75 cases) were alive at the end of five years. When the disease was confined to the breast and axilla, 29.3 per cent (of 66 cases) were alive at the end of five years. When the disease was advanced or inoperable, 23.6 per cent (of 60 cases) were alive at the end of five years. These results compare favorably with results reported when surgery alone has been employed. Keynes has not been completely satisfied with his results, for of late he has added a simple mastectomy to the procedure. He has set down the following rules: (1) Local removal of the tumor if it is large, or the diagnosis is uncertain, followed by radium. (2) Local removal of the breast if the tumor is very bulky, followed by radium. (3) Never dissect the axilla. (4) Radium by itself may be used; (a) if the tumor is of moderate size and the diagnosis certain; (b) if the patient refuses operation. Our experience with these two methods is so limited that we may not express an opinion.

Irradiation before operation has long been advocated but slowly adopted. Its proponents believe that it will reduce the size of the growth, attenuate or kill the scattered cancer cells that may lie in the periphery, and seal off the lymphatics. Thus the operation will become less dangerous in that there is less chance of traumatic emboli with metastasis, or implantation of cancer cells in the wound. If such irradiation is given thoroughly and sufficiently, several weeks to months will be used up in order: (a) To obtain proper effect; and (b) to allow damaged skin to recover. It is also said to interfere with wound repair. Only a few of our cases have had preoperative roentgenotherapy, and in these wound repair was satisfactory.

Postoperative irradiation has a more enthusiastic following. Many believe that the average duration of life has been lengthened. Hermet,[8] of the Curie Institute in Paris, in a recent article concludes: "We are obliged to state that the question of radiotherapy in cancer of the breast is still long distant from having reached the state comparable to that of radiotherapy of other neoplastic localization. The competition between surgery alone, radiation alone, or their combination remains open. Much work is still necessary before arrival at definite opinions. But we know already that irradiation in a considerable number of cases has increased the prognosis of life."

Westermark,[9] in 1930, reported a series of cases treated in 1921–1923 at the Radiumhemmet. After operation, a series of treatments were given to the operative field and the regional nodes including the internal mammary. For the operative field he used a soft filter of 4 Mm., Al. at 50 cm. distance. In the axilla he used 0.5 Mm. Cu. plus 1 Mm. Al. at 40 cm. distance, each dose ⅓ to ¼ S.E.D. A total of 10 to 14 treatments were given in two to three weeks. Seventy-five cases were treated by this method. The surgery had been performed by 19 different surgeons. Thirty-seven per cent were alive and free from recurrence at the end of five years. Twenty-five per cent of the 75 had local recurrences. Forty-five patients had preoperative roentgenotherapy in addition. The preoperative therapy consisted of two tangential

treatments to the tumor with a filter of 0.5 Mm. Cu. and 1 Mm. Al. giving ⅓ S.E.D. If axillary nodes were palpable treatment was also given to them. Of the 45 thus treated 50 per cent were alive and free from recurrence at the end of five years, while eight, or 17 per cent, had local recurrences. He concludes: "It will be clear, therefore, that a five years healing result occurs at a higher rate and that the recurrences are less frequent after a combined surgical and radiologic treatment than after surgical treatment only. This improvement in results is very much in evidence after the pre- and postoperative radiologic treatment."

Wintz,[10] in 1931, has reported on the employment of roentgenotherapy alone. He had 76 per cent five-year cures in 25 cases of Steinthal Group I, and 46.5 per cent five-year cures in 22 cases of Steinthal Group II. Anschutz and Siemens,[11] in 1933, reported equally good results with or without postoperative irradiation in a small series (21) of Steinthal Group I cases. But in the Steinthal Group II, they found, in a large group of cases (292), that postoperative irradiation increased their five-year period of freedom from known recurrence 19.6 per cent.

Webster,[12] in 1936, observes that: "The tissue dose which has been advised in the treatment of mammary cancer has varied from 700 to 8,000 R. The upper limit of tissue dose advised for breast cancer has been that of Lee and Pack who advanced the view in 1931 that 12 or 13 erythema doses may be advisable. Their erythema was a minimal or threshold erythema, and their dose was obtained by a combined roentgen and interstitial radium technique." Webster uses 2,000 to 4,000 R. for simple fractional roentgenotherapy. He treated 182 cases, three-fourths of whom belonged to the Steinthal Group II, the operations having been performed by many different surgeons, with the inevitable variation in surgical technic. With postoperative irradiation he had 42 per cent alive and free from recurrence at the end of five years.

Sufficient reference has been offered to indicate that conservative roentgenologists have had sufficient experience to confirm their belief in the benefit of postoperative irradiation. It must be borne in mind that these reports, of necessity, are based upon the roentgenotherapy of various periods up to 1930. One must also remember that the present attitude of the roentgenologist is that the therapy of that period is archaic. We are told that the present treatment of three to four weeks of fractional doses with a summation of 7,000 to 8,000 R. is the only proper method. To a surgeon, it seems to indicate a lack of faith on the part of the roentgenologists in the results that were obtained five or six years ago. Yet, at that time they were most enthusiastic. It had one unfortunate effect upon many surgeons. It made them content with inadequate surgery. They felt that irradiation would prevent any further trouble.

At present, irradiation is given at the most, to the breast area, and the regional nodes. A few also irradiate the mediastinum. But even if the conclusions of the most enthusiastic roentgenologists are accepted and treatment

is instituted both before and after surgery, or confined solely to irradiation, what are we to do regarding the treatment of cancer that appears elsewhere. It will be noted in Table II that the first site of recurrence was recorded in 119 cases of our series. In over one-half, the recurrence was noted outside of the field of irradiation. Shall we suggest in the future that more fields be irradiated, to include the lungs, the other breast, the vertebrae and pelvis, the abdomen? Such a course presents many serious difficulties. Is it possible or feasible? Or should we rest content with local surgery and roentgenotherapy?

TABLE IV

COMPARISON OF TWO ROOSEVELT HOSPITAL REPORTS

Five-Year Results

	1927[2]	1937
No. operated upon and followed-up.	157	191
Operative deaths	5	5
Alive and well	58 (36%)	55 (27%)
No. without axillary metastases	55	70
Alive and well	39 (70%)	41 (58%)
No. with axillary metastases	97	116
Alive and well	19 (19%)	14 (12%)
No. with known site of recurrence	88	119
Local recurrence	32 (36%)	46 (39%)
Cases not followed five years	56	29

In the series herewith reported, roentgenotherapy was given after operation. The treatment, up to 1930, was based upon the skin reaction and was usually given in massive doses in one or two treatments to a portal and lasted for 30 minutes to an hour. A 200 kilovolt machine was used, with 5 M.A., at a distance of 50 cm. and a filter of .50 Mm. Cu., and 1 Mm. Al. The skin reaction was a severe erythema. This treatment was repeated in two months and again in six months. In a small percentage this was not completed for various reasons.

CONCLUSIONS

We believe that the Halsted type of operation is indicated, except in the matter of skin removal. We have been content to remove a minimum width of five inches of skin in early cases with small tumors, to be followed by the wide subcutaneous dissection as advocated by Sampson Handley. Except in small breasts, we are usually able to effect a plastic closure of the skin. When the tumor is large, more skin must be removed followed by immediate Thiersch skin graft. Our percentage of local recurrence is high, but no higher than those given in the reports from Johns Hopkins Hospital, where the typical Halsted operation is performed.

In 50 cases with known sites of recurrence, in which the Halsted-Handley operation had been performed, there were no axillary recurrences. In 69 cases with known sites of recurrence, in which the pectoralis minor was allowed to remain, there were six local, axillary recurrences.

Surgery gives a five-year prognosis of freedom from recurrence, that varies with the age of the patient, the type of cancer, and the stage of the disease.

In our cases without axillary metastases, roentgenotherapy has not increased the prognosis of five-year freedom from recurrence, although it has given to a small percentage the opportunity to live longer.

In our cases with axillary metastases, roentgenotherapy has unquestionably given a 10 per cent increase in the prognosis of five-year freedom from disease.

It is true that this report is a disappointment to us, in that our percentage results are not as good as those reported by the writer[2] in 1927. We had hoped to show improvement with the added routine use of the roentgenotherapy then in vogue. Our only possible explanation may be that our pathologic, diagnostic ability is better, and that our cases are more carefully studied and followed up.

In the hope of improvement, during the past three years, we have changed our method to the fractional dose treatment. With this improved method, it is hoped that the incidence of local recurrence may be reduced, and their appearance delayed. Still a real problem is that of regional and distant metastases that are present at the time of operation, although unrecognized. An encouraging approach to this has been made in the sterilization of the patient. I am inclined to doubt the advisability of treating other endocrine glands.

REFERENCES

[1] Peck, Charles H., and White, William Crawford: Tumors of the Breast. ANNALS OF SURGERY, **75,** 641–651, June, 1922.

[2] White, William Crawford: Late Results of Operation for Carcinoma of the Breast. ANNALS OF SURGERY, **86,** 695–701, November, 1927.

[3] Halsted, W. S.: ANNALS OF SURGERY, **20,** 507, 1894.

[4] Meyer, Willy: Med. Rec., **46,** 746, December 15, 1894.

[5] Handley, W. Sampson: Cancer of the Breast. 2nd Edit., London, 1922.

[6] Lewis, Dean, and Rienhoff, Jr., William Francis: A Study of the Results of Operation for the Cure of Cancer of the Breast. ANNALS OF SURGERY, **95,** 336–400, March, 1932.

[7] Keynes, Geoffrey: The Radium Treatment of Carcinoma of the Breast. Brit. Jour. Surg., **19,** 415–480, 1932.

[8] Hermet, P.: La radiotherapie dans le traitment du cancer du sein. Paris Med., **1,** 233–242, 1936.

[9] Westermark, N.: Result of Combined Surgical and Radiological Treatment of Cancer Mammae at Radiumhemmet, 1921–1923. Acta Radiol., **11,** 1–32, 1930.

[10] Wintz, H.: Ergebnisse der Roentgentherapie des Mammakarzinome. Deutsch. med. Wchnschr., **57,** 1569, 1931.

[11] Anschutz, W., and Siemens, W.: Uber die Ergolge der Nachbestrahlung radikal operierter Mammakarzinome. Zentralbl. f. Chir., **60,** 923–930, 1933.

[12] Webster, J. H. D.: Cancer of the Breast; X-Ray and Radium Treatment of Operable and Borderline Cases. Brit. Jour. Radiol., **9,** 463–466, 1936.

[13] Keynes, Geoffrey: The Place of Radium in the Treatment of Cancer of the Breast. ANNALS OF SURGERY, **106,** 619–630, October, 1937.

DISCUSSION: DR. HUGH AUCHINCLOSS (New York) said that he did not wish to discuss statistics in these cases as they are usually compiled. When he first began studying cancer of the breast he was convinced that statistics would tell the story. Indeed they might, if the many variants, qualitative and quantitative, of the individual cases could be taken into account. After 25 years, it has become perfectly clear that this has not been done and that with

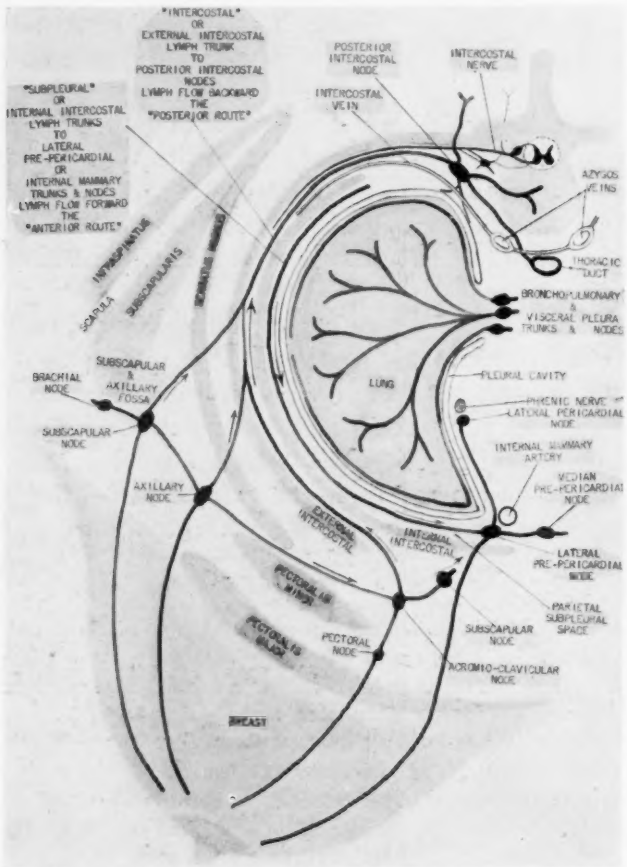

FIG. 1.—Diagram to show the lymphatic drainage posteriorly, along the intercostal lymph trunks to the posterior intercostal nodes, and anteriorly, to the anterior mediastinum where the prepericardial nodes near the internal mammary vessels are situated. The posterior intercostal lymphatics are situated in the so-called "azygos route." The anterior mediastinal lymphatics are situated in the subpleural tissues and along the internal mammary vessels, phrenic nerve, diaphragm, liver and round ligament. (This drawing is a modification of Fig. 10, p. 21, from "Cancer of the Breast Clinically Considered," by Cecil H. Leaf, one of the most thoughtful treatises on cancer of the breast that has ever been published.)

our present knowledge it is quite impossible. A few of the conclusions drawn from them are truths that have been known for many years. Most of the rest of them are false.

He wished to make it quite clear that he believed he had seen benefits derived from irradiation of breast cancer, but also should like to make it clear that, in spite of these benefits, he had never seen a case proven rid of the disease by the employment of roentgenotherapy alone.

Experienced clinical observers vary widely in their descriptions of the same case. Often one or two miscroscopic sections, following radiation, have been taken from a place that had been considered typical, yet no cancer was found. If many sections are made from the surrounding areas, however, cancer has always been discovered, or the clinical course has demonstrated that it had not been eradicated.

Hope still exists that irradiation may be made more effective than it is to-day, not only in collaboration with surgery, but that it may even replace surgery. It certainly has not done so as yet, and those who assume that it has, may cause women to lose their lives unnecessarily.

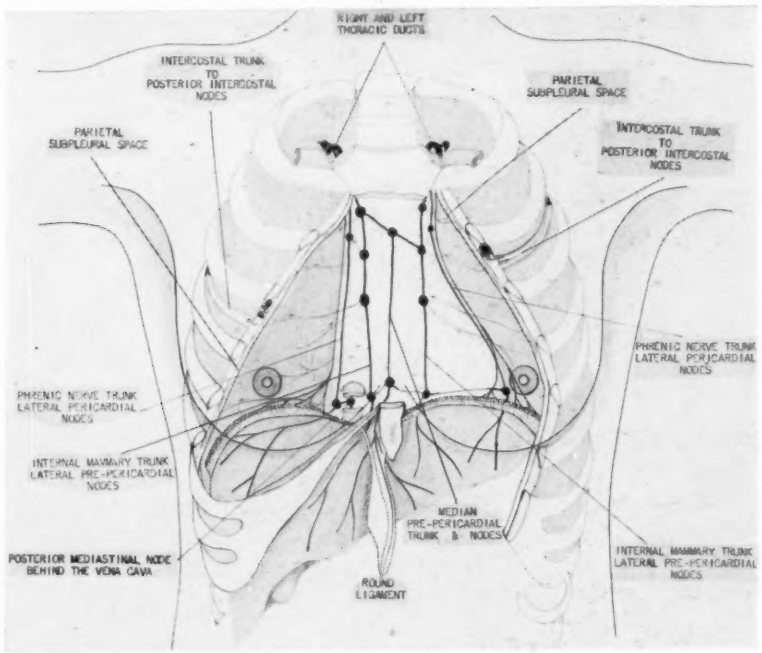
RIGHT AND LEFT THORACIC DUCTS
INTERCOSTAL TRUNK TO POSTERIOR INTERCOSTAL NODES
PARIETAL SUBPLEURAL SPACE
PARIETAL SUBPLEURAL SPACE
INTERCOSTAL TRUNK TO POSTERIOR INTERCOSTAL NODES
PHRENIC NERVE TRUNK LATERAL PERICARDIAL NODES
PHRENIC NERVE TRUNK LATERAL PERICARDIAL NODES
INTERNAL MAMMARY TRUNK LATERAL PRE-PERICARDIAL NODES
MEDIAN PRE-PERICARDIAL TRUNK & NODES
POSTERIOR MEDIASTINAL NODE BEHIND THE VENA CAVA
ROUND LIGAMENT
INTERNAL MAMMARY TRUNK LATERAL PRE-PERICARDIAL NODES

FIG. 2.—Diagram to show the lymphatic drainage of the inner portion of the breast, the subpleural spaces, the diaphragm, the liver, and the round ligament, to the anterior mediastinal lymph nodes.

We must not talk about "axillary node involvement" or "uninvolvement" based upon a clinical examination alone. It is a sure sign of ignorance, if we do. Very large, hard, and even visible masses of nodes have been called metastases, when they were not, and nodes containing cancer have often been overlooked on the physical examination. There is definite proof of this in our records.

One subject has been touched upon that is rather close to our minds and hearts in the study of this disease. Are we irradiating breast cases intelligently? Are we irradiating the tissues that should be irradiated? After all, do we know what tissues to irradiate, for if we do not, what is the use of irradiating at all? If we radiate the breast tumor, or the axilla, we are radiating tissues that, surgically, it has been pretty well demonstrated can be removed successfully. He could think, perhaps, of only one or two cases of "persistences" in the subclavian, axillary and brachial nodes in all the cases that he had personally followed. Occasionally, particularly in advanced cases, there may be a metastasis in the skin in the neighborhood of the excision. But even this

is a relatively rare site for metastases to occur, in our experience. If the skin be widely removed, as Doctor Halsted recommended, and wide undermining,

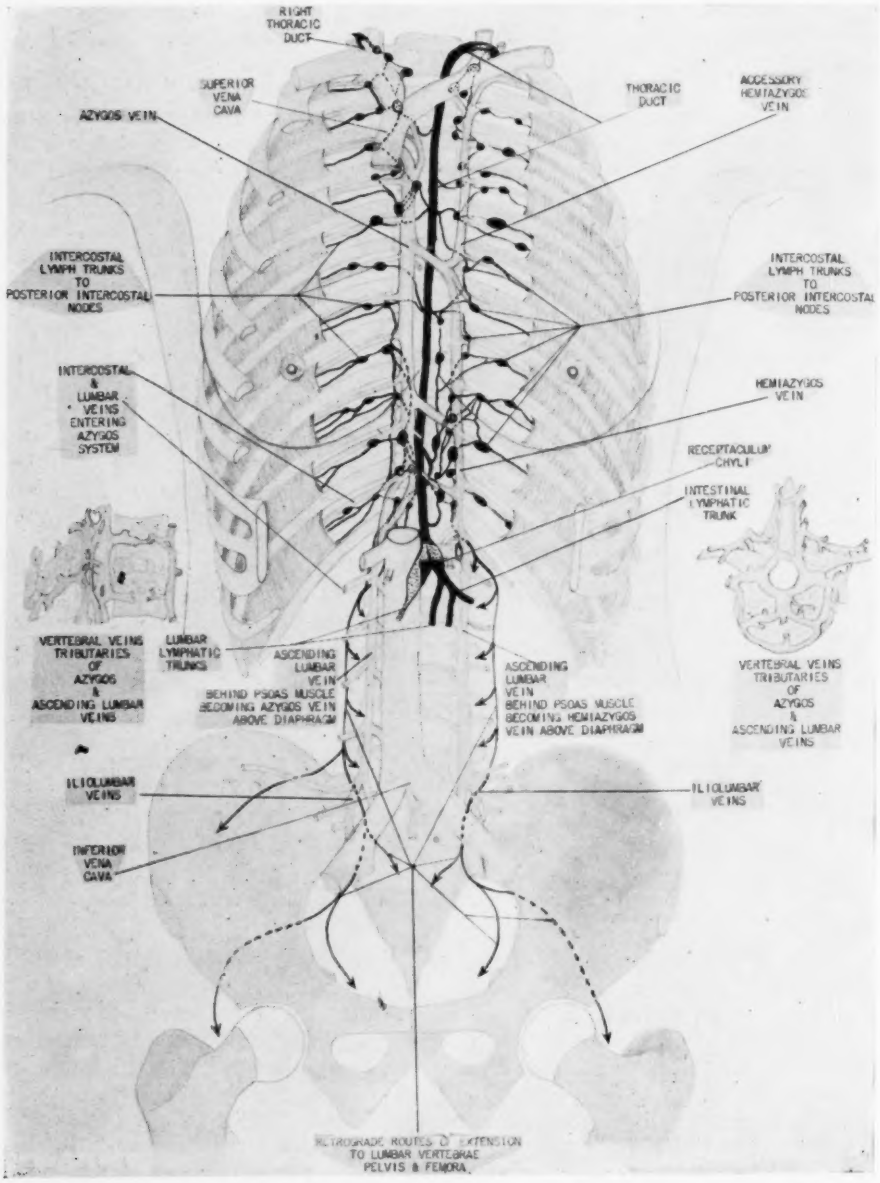
RIGHT THORACIC DUCT
SUPERIOR VENA CAVA
AZYGOS VEIN
THORACIC DUCT
ACCESSORY HEMIAZYGOS VEIN
INTERCOSTAL LYMPH TRUNKS TO POSTERIOR INTERCOSTAL NODES
INTERCOSTAL LYMPH TRUNKS TO POSTERIOR INTERCOSTAL NODES
INTERCOSTAL & LUMBAR VEINS ENTERING AZYGOS SYSTEM
HEMIAZYGOS VEIN
RECEPTACULUM CHYLI
INTESTINAL LYMPHATIC TRUNK
VERTEBRAL VEINS TRIBUTARIES OF AZYGOS & ASCENDING LUMBAR VEINS
LUMBAR LYMPHATIC TRUNKS
ASCENDING LUMBAR VEIN BEHIND PSOAS MUSCLE BECOMING AZYGOS VEIN ABOVE DIAPHRAGM
ASCENDING LUMBAR VEIN BEHIND PSOAS MUSCLE BECOMING HEMIAZYGOS VEIN ABOVE DIAPHRAGM
VERTEBRAL VEINS TRIBUTARIES OF AZYGOS & ASCENDING LUMBAR VEINS
ILIOLUMBAR VEINS
ILIOLUMBAR VEINS
INFERIOR VENA CAVA
RETROGRADE ROUTES OF EXTENSION TO LUMBAR VERTEBRAE PELVIS & FEMORA

FIG. 3.—Diagram illustrating the so-called "azygos route." The term "azygos route" is intended for the continuous fascial planes, in which are situated the tributaries of the azygos system of veins. In these fascial planes are the lymphatics that drain posteriorly a greater part of the breast. It offers an explanation for the metastases from cancer of the breast to the intercostal spaces, to the posterior mediastinum, to the vertebral bodies, to the pelvic bones and retroperitoneal tissues of the pelvis and the femora.

close to the skin, be effected, skin metastases are rare. The real trouble lies elsewhere. It lies in those places to which the disease has spread beyond the

tissues capable of being removed by operation. If radiation is to be of value in the treatment of breast cancer, it should be especially directed to those tissues incapable of being removed by operation. These tissues serve as secondary distributing foci.

Most radiation at the present time, indeed practically all of it, is being given to the tissues capable of being removed at operation. It is perfectly true that in doing this certain conducting paths to these quasi "secondary distributing foci" may be radiated. This may be of help, but, if the other secondary distributing foci themselves are not radiated, this effort can be of but little more than temporary value. The important feature of the whole subject, therefore, is to understand where these so-called "secondary distributing foci" exist, so that intelligent radiation may be directed toward them.

The local, so-called recurrences, but better "persistences," of the disease are often thought to be skin persistences when they are not. They are more likely to be persistences of the disease in the intercostal tissues of the anterior or lateral chest wall.

Emphasis has been laid for many years upon the extensions to the so-called supraclavicular lymph nodes. It is true that extensions to the lymphatics above the clavicle occur. As a matter of fact, however, these extensions constitute only a part, and, indeed, rather a lesser part of those extensions that actually cause the death of the patient. The routes along which the disease more often spreads are: To the pleura, anterior mediastinum, vertebral column, liver, pelvic bones, and to the upper portions of the femora; in dramatic contrast to the extraordinarily rare distribution to the lower arms, legs, hands and feet. They are not generally understood. The observations of Sampson Handley and, in particular, those of Cecil H. Leaf, published after his death by Cecil Rowntree in 1912, have been noteworthy contributions on this subject. The accompanying diagrams are offered in the hope that they will present an explanation of the fascial planes that determine, and include, the lymphatic routes along which this disease preeminently spreads to these, above mentioned, tissues.

Extension along the channels indicated in the illustrations presupposes the understanding of dissemination of disease by so-called "circuitous" and "retrograde" routes, now generally recognized and accepted by pathologists.

It is hoped that these diagrams may aid in determining more reasonable and logical areas for helpful radiation in breast cancer.

PEPTIC ULCER

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THE present study comprises an analysis of 520 cases of peptic ulcer seen at the Presbyterian Hospital, New York. In each instance the lesion was seen or the diagnosis confirmed by the surgeon or the pathologist. Such a group of so-called "proven ulcers" was examined in detail in order, insofar as possible, to correlate the clinical, pathologic and roentgenologic findings, and to compare these with the observations of other investigators.

It was about a century ago that Cruvelhier (Bevan[16]) first distinguished gastric ulcer from carcinoma, and a few years later Quincke (Bevan[16]) named the condition "peptic ulcer." Since then an enormous literature has accumulated, attesting to the interest in the subject. Many of the statistics are, however, at variance, due at least in part to the variety of sources drawn upon for information. Thus the pathologist, the roentgenologist, the surgeon and the internist have each approached the subject from a different aspect, often with very little agreement in their results (Albrecht[5] and Boyd[21]). Variations may be noted from time to time and from place to place, and in the light of more recent knowledge some textbook statistics are subject to question. Robinson,[125] in an interesting review, has pointed out many of these discrepancies.

Etiology.—It is not within the scope of this paper to discuss in detail the many theories advanced to explain the occurrence and chronicity of peptic ulcers. With the exact etiology unknown, certain factors which probably influence the development of such lesions are suggested in the hypotheses offered. It is quite generally felt that the destructive action of the gastric juice brings about a condition which is unique in its chronicity, as well as in its localization in a mucosa which is bathed by such secretions. To this latter fact the presence of peptic ulcers in the esophagus, Meckel's diverticulum and at the site of a gastro-enterostomy is offered as evidence, inasmuch as ectopic gastric mucosa has been frequently found in these situations when so involved (Lindau and Wulff,[93] and Matthews and Dragstedt[100]). Gastric hyperacidity or the failure of regurgitation of alkaline duodenal secretions to reduce gastric acidity have been advanced as the primary initiating factor by many, including Sippy,[136] Bolton,[20] Boyd,[21] Chace,[28] Deaver and Burden,[40] Dragstedt,[45] Hurst and Stewart,[76] Karsner,[82] Martin and Burdin,[98] Matthews,[99] Mann,[97] Miller,[103] Morton,[106] Moszkowicz,[107] Osler,[111] Robinson,[126] Somervell and Orr,[139] and Rivers.[121]

Deficiencies of blood supply and vascular "accidents" have been discussed as etiologic factors by many others (Bevan,[16] Eggers,[46] Wilbur in Euster-

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man and Balfour,[52] Friedenwald and Love,[57] Gaither,[59] Hauser,[68] Karsner,[82] Mayo,[101] Miller,[103] Osler,[111] Payr,[114] Reeves,[118] Robinson,[125] Smithies,[137] Somervell and Orr,[139] Virchow,[149] and Wilkie[153]).

The presence of ulcers in nervous, so-called dynamic individuals and particularly in association with periods of stress, fatigue and emotional tension, has been described with increasing frequency in recent writings. In this connection Robinson's[125] article is of interest. Among the large group who have expressed views on this subject are Bevan,[16] Blahd,[18] Brown in Cecil,[27] Chace,[28] Wilbur in Eusterman and Balfour,[52] Hunt,[74] Hurst and Stewart,[76] Kaiser,[81] Osler,[111] Rivers,[121] Robinson,[126] Smithies,[137] and Somervell and Orr.[139]

Interesting indeed is the possibility of the association of the mental state with changes in the gastric vascular bed. An imbalance in the vegetative nervous system has been considered to be an etiologic factor by Beaver and Mann,[13] Cushing,[38] Hartzell,[66] Singer[134] and Von Bergmann,[150] among others.

Some authors concede that trauma may give rise to peptic ulcers, in certain situations, either by direct violence to the abdomen, or as might occur with certain harsh foods or foreign substances in the diet (Robinson,[125] Brown in Cecil,[27] Rivers,[121] Blahd,[18] Boyd,[21] Osler,[111] and Somervell and Orr,[139] Gallagher[60]).

Other etiologic factors which have been suggested include focal infections (Bevan,[16] Brown in Cecil,[27] Wilbur in Eusterman and Balfour,[52] Hunt,[74] Meisser,[102] Miller,[103] Osler,[111] and Robinson[125]), and a local infection such as a gastritis or duodenitis (Boyd,[21] Wilbur in Eusterman and Balfour,[52] Konjetzny,[88] Orator and Metzler,[110] Puhl,[117] Karsner,[82] Osler,[111] Rosenow,[128] and Smithies[137]). A diffuse inflammation of the stomach or duodenum has been noted to be a frequent precursor of ulcer by Bevan,[16] Blahd,[18] Smithies,[137] and Wilbur in Eusterman and Balfour.[52]

Alcohol and tobacco have been seriously considered as a cause of ulceration, but probably have more to do with the maintenance of the condition (Bevan[16] and Hunt[74]). Somervell and Orr[139] studied a large group of Indian natives and believed a vitamin deficiency to be the inciting agency. Endocrine disturbances are mentioned by Karsner[82] and Chace.[28] Curling,[37] in 1842, pointed out the association of ulceration of the duodenum with severe burns. Cushing[38] pointed out the association of peptic ulceration with disease, particularly with tumors involving the midbrain. Diseases of the abdominal viscera other than the stomach and duodenum, and particularly the gallbladder and the appendix, have been thought to have some bearing on the subsequent formation of peptic ulcers (Aaron,[1] Braithwaite,[22] Hunt,[74] Osler,[111] Miller[103]). The presence of aberrant Brunner's glands in the gastric mucosa in association with ulcer was reported by Corria,[35] who felt that this might be an etiologic factor.

In summary, it would seem that peptic ulcers arise in an area of mucosa in which the resistance has been lowered by any one or several processes. This area is bathed by gastric juice, which may well increase the smallest injury, and in some manner contribute to the chronicity of the disease.

Frequency and Distribution.—Peptic ulcer patients are apparently increasing in number from year to year (Boyd,[21] Chace,[28] Hinton,[70] and Wilkie[152]). The general population apparently suffers from this malady in percentages which are quite consistently in the values of 1.5:2.0 per cent (Brown in Cecil,[27] Osler,[111] and Sturdevant and Shapiro[146]). Somervell and Orr,[139] in India, estimated the frequency to be much lower (0.5 per cent), indicating the variability associated with different places of study. Hurst and Stewart[76] considered as many as 10 per cent of the population to be sufferers.

In hospital admissions the frequency is in about the same ratio (1.1:1.8 per cent) (Emery and Monroe,[48] Goldman,[64] Lynch[94]). Table I analyzes the ratio-incidence among 118,878 admissions to the Presbyterian Hospital, New York, in the years 1924–1934, inclusive. Of these, 1,829 were for peptic ulcer, or about 1.5 per cent. These ulcer admissions were found to represent 1,460 patients, suggesting the frequency with which such patients make more than one visit for treatment

TABLE I

ANALYSIS OF THE INCIDENCE OF PEPTIC ULCER AMONG GENERAL HOSPITAL ADMISSIONS

(*Presbyterian Hospital, 1924–1934, incl.*)

Total hospital admissions 1924–1934, incl.		118,878	
Total admissions for ulcer 1924–1934, incl.		1,829	1.5%
Gastric ulcer	464		
Duodenal ulcer	1,365		
Total patients admitted for ulcer 1924–1934, incl.		1,460	
Gastric ulcer	384		
Duodenal ulcer	1,076		
Total number of autopsies studied		2,395	
Total cases showing ulcer		174	7.2%
Sex distribution—average for four months (all hospital admissions—about 3,600 patients):			
Males			51.5%
Females			48.5%
Race distribution—average for four months (all hospital admissions—about 3,600 patients):			
White			88 %
Black			11.5%
Other			0.5%

In a study of autopsy material the frequency is usually higher. Sturdevant and Shapiro[146] noted that 2 per cent of 7,700 postmortem examinations revealed peptic ulcers. Cleland,[32] in 1,000 consecutive autopsies, found 18 gastric and 11 duodenal ulcers, or a total percentage of 2.9. Hurst and Stewart[76] report 5.75 per cent, and Bevan[16] quotes the studies of Hart in Berlin who discovered some evidence of ulceration in 10 to 12 per cent of all autopsies. Robertson and Hargis[124] and Stewart[142] give similar statistics. In 1,000 autopsies Lehmann[92] found 20.2 per cent to have either a frank ulcer or a scar. Table I shows that among 2,395 consecutive autopsies studied, 174, or 7.2 per cent, showed peptic ulcers.

From other sources an even higher incidence is reported. For example,

a group of "dyspeptics" yielded 24 per cent positive cases in a series studied by Albrecht.[5] Suspected cases studied roentgenologically by Kirklin in Eusterman and Balfour[52] revealed 13.1 per cent to have ulcers, and in a group of similar cases Percy and Beilin[115] found 19.2 per cent.

Sex Distribution.—In all of the reports studied the predominance of male patients in both the gastric and duodenal ulcer groups is apparent (Boyd,[21] Moynihan,[109] and Osler[111]). In those summaries which group all cases as "peptic ulcers," the ratio of males to females varies from 1.1:1.0 to 4.8:1.0 (Blackford and Dwyer,[17] Brown in Cecil,[27] DeLario,[41] Eusterman and Balfour,[52] Lynch,[94] Smithies,[138] Stewart[142] and Wilkie[152]). In gastric ulcer patients, most authors find the predominance of men less marked than in the duodenal group (Chang,[29] Cleland,[32] Emery and Monroe,[48] Jordan,[79] Lynch,[94] Percy and Beilin,[115] Roof[127] and Wilkie[152]). Quite in contrast, however, are the reports of Hinton and Trabek,[69] who found the ratio to be 7.7:1.0 for gastric ulcers, and of Miller, Pendergrass and Andrews,[104] whose ratio in this group was 6.6:1.0.

Table II shows the sex distribution and type of ulcers in the series, and indicates that the ratios of males to females were about the average of the above cited reports. The ratio for all of the cases is 3.6:1; 3.7:1 in the gastric, and 3.5:1 in the duodenal cases. Little variation is noted between the medical pathology, surgical pathology and celiotomy cases. A four month survey of all admissions to the Presbyterian Hospital showed the distribution of the sexes to be essentially equal (Table I).

Racial Distribution.—Table II shows the racial distribution in this group of patients, which when compared with the four month survey of admissions, as indicated in Table I, would seem to show no racial selectivity. Many reports show a tendency for the white race to be more frequently the victim of peptic ulcer (DeLario,[41] Lynch[94] and Roof[127]). Robinson[126] quoted many figures from different parts of the world to support his conclusion that peptic ulcer is "found only among susceptible individuals of the white race."

Age Incidence.—The age of the patients is analyzed in Table II, with the youngest and the oldest in each classification. These statistics would seem to indicate that gastric ulcer patients were slightly older than those with duodenal ulcer (49.5 and 44 years, respectively). This is in accord with the conclusions of Blackford and Dwyer,[17] Eusterman and Balfour,[52] Hunt,[74] Miller, Pendergrass and Andrews,[104] Percy and Beilin[115] and Roof.[127] The average age in each type of ulcer corresponds closely to that given as most frequent by the majority of writers (Blackford and Dwyer,[17] Boyd,[21] Chang,[29] Cleland,[32] DeLario,[41] Emery and Monroe,[48] Eusterman and Balfour,[52] Hinton and Trabek,[69] Hunt,[74] Jordan,[79] Lynch,[94] Miller, Pendergrass and Andrews,[104] Osler,[111] Percy and Beilin,[115] Robertson and Hargis,[124] Roof[127] and Smithies[138]). Percy and Beilin[115] give 69 as the age of their oldest gastric ulcer patient and 15 as that of the youngest. Of their duodenal cases, the oldest and youngest were 74 and 18, respectively.

Location.—In allocating ulcers to the stomach or to the duodenum the

TABLE II

ANALYSIS OF TYPES OF ULCER, SEX AND RACIAL DISTRIBUTION, AND AGE INCIDENCE

	—Medical Pathology—			——Surgical Pathology——			————Celiotomy————			————————Totals————————		
	G.U.	D.U.	Comb.	G.U.	D.U.	Comb.	G.U.	D.U.	Comb.	G.U.	D.U.	Comb.
Total patients....	76	79	19	44	60	11	38	180	13	158	319	43
Total ulcers.....	95	98	19	55	71	11	51	193	13	201	362	43
Male.....	77 (81%)	78 (80%)		40 (73%)	47 (66%)		41 (80%)	156 (81%)		158 (79%)	281 (78%)	
Female.....	18 (19%)	20 (20%)		15 (27%)	24 (34%)		10 (20%)	37 (19%)		43 (21%)	81 (22%)	
White.....	84 (88%)	90 (92%)		50 (91%)	69 (97%)		46 (90%)	176 (91%)		180 (89%)	335 (92%)	
Black.....	9 (10%)	5 (5%)		4 (7%)	1 (1%)		3 (6%)	11 (6%)		16 (8%)	17 (5%)	
Yellow.....	2 (2%)	3 (3%)		1 (2%)	1 (1%)		2 (4%)	6 (3%)		5 (3%)	10 (3%)	
Average age at autopsy or admission.....	53.6 yrs.	50.8 yrs.		47.2 yrs.	43.6 yrs.		44.2 yrs.	40.8 yrs.		49.5 yrs.	44.0 yrs.	
Youngest.....	18	4		23	12		23	20		18	4	
Oldest.....	76	83		78	67		72	68		78	83	

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marked predominance of the latter site is seen in all but the postmortem series (Table II). A review of the literature shows the ratio of duodenal ulcers to gastric ulcers to vary from 1.1 :1 to 9.5 :1 for clinical reports (Albrecht,[5] Balfour,[9] Blackford and Dwyer,[17] DeLario,[41] Hinton and Trabek,[69] Eggleston,[47] Emery and Monroe,[48] Hinton,[70] Holmes and Schatzki,[73] Hurst and Stewart,[76] Lynch,[94] Miller, Pendergrass and Andrews,[104] Osler,[111] Percy and Beilin,[115] Roof,[127] Smithies,[138] Sutherland,[147] Walton[151] and Wilkie[153]). In the series herewith reported the ratio of duodenal to gastric ulcers is about 2:1. In contrast, Boyd[21] states that gastric ulcers are probably more frequent than duodenal, and several reports obtained from an analysis of pathologic material would tend to agree (Cleland,[32] Eusterman and Balfour,[52] Lehmann,[92] Robertson and Hargis,[124] and Sturdevant and Shapiro[146]). However, Stewart,[142] reporting postmortem findings found duodenal to exceed gastric ulcers. The possibility arises that the inclusion of acute ulcers by the pathologist may be an explanation for this discrepancy. In this series such ulcers appear almost exclusively in the autopsied group (Table III), and are more frequent in the stomach than in the duodenum. It may be that some of these are of the type of "erosions" described, among others, by Steinberg.[141]

Combined ulcers, that is, ulcers in both the stomach and duodenum in the same patient, were present in 8 per cent of the cases. This is higher than in the report by Albrecht,[5] with a frequency of 1.7 per cent, by Emery and Monroe,[48] with 1.8 per cent, or in those by DeLario[41] and Sturdevant and Shapiro,[146] with 3 per cent each. Kirklin in Eusterman and Balfour[52] found 15 per cent of gastric ulcers to be associated with duodenal ulcers and 6 per cent of duodenal ulcers to be associated with gastric ulcers. Carman[25] concurs with the former figure and Walton[151] with the latter. Wilkie's[152] 51 combined ulcers in 362 peptic ulcer patients would indicate a frequency of 15 per cent. Rivers[122] found a coexistence of 13 per cent.

Pathology.—Pathologically, gastric and duodenal ulcers may be considered together, as the lesions are essentially similar. Each occurs in a portion of the digestive tract which has much in common with the other, both embryologically and anatomically. The stomach and duodenal bulb arise from the foregut, both have blood supply from the celiac axis, and each is bathed by acid gastric secretion (Boyd[21]). The majority of peptic ulcers are located in the gastric *"magenstrasse"* and the bulb, and here the vascularity has the common characteristic of an "end-artery type," with a paucity of capillaries (Bevan,[16] Reeves,[118] Wilkie[153]).

By far the greatest number of benign gastric ulcers are seen in a limited area of the lesser curvature. As shown in Table III, 77 per cent of the gastric ulcers were situated on the lesser curvature in the area of the *"magenstrasse"* (gastric pathway). The anterior wall of the stomach was the next most frequent site. Sproull,[140a] in 1931, analyzed all of the reported cases of gastric ulcer occurring on the greater curvature of the stomach, and concluded that benign ulcers in this site are extremely rare. In the Presbyterian Hospital series this was also found to be the case. Two and one-half per

cent of the gastric ulcers were found to be on the greater curvature. This represents four cases, all of which were "acute ulcers" noted at postmortem. Not a single instance of chronic, benign ulcer of the greater curvature was found. Welch (Osler[111]) reported the lesser curvature to be only slightly more frequently involved than the posterior wall. This is at variance with the usual opinion of other observers, which agrees more closely with the results indicated in this series (Bevan,[16] Boyd,[21] Eusterman and Balfour,[52] Holmes and Hampton,[72] Karsner,[82] Köhler,[87] Miller, Pendergrass and Andrews,[104] Rivers,[121] Roof[127] and Stewart[143]).

In actual contact with the pylorus, or definitely involving it in the process of the disease, there were 13 per cent of the gastric ulcers in the cases here reported. DeLario[41] found 4 per cent at this site, but in most series the only statement is "near the pylorus," making comparisons with this finding difficult. The farthest from the pylorus of any gastric lesion was found to be 18 cm., as measured on the specimen resected at operation, while the average distance for this group was 3.9 cm. This is the region just distal to the incisura, and is the site of predilection noted by most authors (Boyd,[21] Chang,[29] Eusterman and Balfour,[52] Hinton and Trabek,[69] Holmes and Schatzki,[73] Karsner,[82] Moynihan,[109] Stewart,[143] Sturdevant and Shapiro[146]).

The average size of the craters of benign gastric ulcers is most frequently stated to be "under 3 cm." Alvarez[6] considers such ulcers rarely to exceed 2.4 cm., and Boyd[21] places the usual upper limit at 2 cm. Chang[29] found 70 per cent of 63 cases to be under 3 cm. in diameter, the smallest measuring but 6 Mm. and the largest, 5 cm. MacCarty in Eusterman and Balfour[52] reports benign ulcer craters varying in size from 1 Mm. to 19 cm., while Robertson in Eusterman and Balfour[52] places the average size of the crater at 1 cm. Sturdevant and Shapiro[146] analyzed 120 cases and recorded an average of 2.35 cm. The average, noted in Table III, for this group is 1.66 cm. with the smallest being considered a healed or microscopic crater and the largest 8 cm. across.

Duodenal ulcers are practically limited to the bulb, very few being noted beyond this portion. Only one of the 287 studied in this series was beyond the distal bulb, it being found in the third portion. Of the remaining cases the most distant was five centimeters, and the average measurement, from the proximal margin, of the entire group was 0.79 cm. Only 11.5 per cent of the cases were in actual contact with the pyloric ring. These findings are consistent with the general opinion of others. Boyd[21] and Köhler[87] place 90 per cent of ulcer craters within the first one and one-half inches of the duodenum. Roof[127] found 73.5 per cent less than 2.5 cm. beyond the pylorus. Jefferson[78] reported that 99.2 per cent of 496 duodenal ulcers were above the ampulla of Vater. In the Presbyterian Hospital series (Table III) about 70 per cent of the duodenal ulcers were on the anterior wall, the remainder being distributed about equally on the posterior and the superior surfaces. Only a small number were on the greater curvature or inferior side. Köhler[87] does

TABLE III

ANALYSIS OF THE LOCATION, SIZE, TYPES, AND RESULTANT DEFORMITY OF ULCERS IN 447 PATIENTS

	Medical Pathology			Surgical Pathology			Celiotomy			Totals		
	G.U.	D.U.	Comb.	G.U.	D.U.	Comb.	G.U.	D.U.	Comb.	G.U.	D.U.	Comb.
Location												
No. of cases giving locus	66	56		53	67		41	164		160	287	
Ant. wall	14 (21%)*	18 (32%)*		18 (34%)*	41 (61%)*		20 (49%)*	140 (85%)*		52 (32%)*	199 (69%)*	
Post. wall	5 (8%)	29 (52%)		12 (23%)	24 (36%)		6 (15%)	16 (10%)		23 (14%)	69 (24%)	
Less. curvat.	60 (91%)	23 (41%)		41 (77%)	15 (22%)		22 (54%)	28 (17%)		123 (77%)	66 (23%)	
Gr. curvat.	4 (6%)	5 (9%)		0 (0%)	1 (2%)		0 (0%)	4 (3%)		4 (2½%)	10 (0.3%)	
Av. distance to pylorus	5.3 cm.	.98 cm.		3.8 cm.	.58 cm.		2.1 cm.	.82 cm.		3.7 cm.	.79 cm.	
Farthest from pylorus	12.0 cm.	5.0 cm.		18.0 cm.	3.0 cm.		16.0 cm.	4.0 (1 in 3rd portion)		18.0 cm.	5.0 cm.	
Involving pylorus	10 (10%)†	2 (2%)†	6 (33%)†	8 (15%)†	10 (14%)†	5 (45%)†	8 (16%)†	20 (10%)†	8 (61%)†	26 (13%)†	42 (12%)†	19 (44%)†
Size												
Av. size crater	1.73 cm.	1.3 cm.		1.6 cm.	.8 cm.					1.66 cm.	1.05 cm.	
Largest crater	7.0 cm.	5.0 cm.		8.0 cm.	4.0 cm.					8.0 cm.	5.0 cm.	
Types												
Acute	46 (48%)†	33 (33%)†		0 (0%)†	0 (0%)†		1 (2%)†	2 (1%)†		47 (23%)†	35 (10%)†	
Chronic	36 (38%)	57 (58%)		48 (87%)	59 (83%)		43 (85%)	179 (96%)		127 (63%)	295 (81%)	
Healed	4 (4%)	8 (8%)		2 (4%)	11 (15%)		4 (8%)	12 (6%)		10 (5%)	31 (9%)	
Carcinoma in old ulcer	9 (9%)	0 (0%)		4 (8%)	1 (2%)		3 (6%)	0 (0%)		16 (8%)	1 (0.3%)	
Scarring												
No. of cases noting scarring	94	88		55	68		45	179		194	335	
None	50 (53%)*	32 (36%)*		0 (0%)*	0 (0%)*		1 (2%)*	2 (1%)*		51 (26%)*	34 (10%)*	
Slight—no deformity	1 (1%)	11 (13%)		3 (5%)	1 (2%)		8 (18%)	23 (13%)		12 (6%)	35 (10%)	
Marked—no deformity	8 (8.5%)	17 (19%)		6 (11%)	15 (22%)		7 (15%)	30 (17%)		21 (11%)	62 (19%)	
Slight—with deformity	7 (7.5%)	2 (2%)		3 (5%)	11 (16%)		7 (15%)	41 (23%)		17 (9%)	54 (16%)	
Marked—with deformity	28 (30%)	26 (30%)		43 (79%)	41 (60%)		22 (50%)	83 (46%)		93 (48%)	150 (45%)	

* Percentage of those giving this data.

† Percentage of all ulcers.

not agree with this, placing the greater portion on the superior aspect of the bulb. Karsner[82] believes the posterior wall is more often involved.

The crater of a duodenal ulcer is usually smaller than that of a gastric lesion, averaging about 1 cm. in this series. Craters were noted as varying from a minute, microscopic ulceration to one with a diameter of 5 cm. Deaver,[39] MacCarty in Eusterman and Balfour,[52] and Sturdevant and Shapiro,[146] all place their cases within this same range.

Table II also indicates the well-known fact that peptic ulcers are frequently multiple. There were 201 gastric ulcers in 158 patients and 362 duodenal ulcers in 319 patients.

As might be expected, the "combined" ulcers more frequently involved the pylorus (Table III), inasmuch as many lay on both the duodenal and gastric side of the muscular ring and were continuous over its surface.

The microscopic picture in peptic ulcer is variable. They may be acute, chronic or healed, and in this series the chronic type was most frequently found. The acute ulcers were more frequent in the gastric group and the healed more numerous in the duodenum (Table III). This has been commented upon above as offering a possible explanation for the lack of agreement of the pathologic and clinical statistics. No comparable reports were found in the literature.

The ulcer crater may be represented by a small, shallow erosion; or penetrate the wall to varying depths; or perforate to an adjacent viscus or into the peritoneal cavity. They are most frequently deep and callous, involving the submucosa and muscularis (Carman,[25] MacCarty in Eusterman and Balfour,[52] Boyd[21]). The margins of the crater may be overhanging, receding or terraced (Boyd,[21] MacCarty in Eusterman and Balfour,[52] Osler,[111] Robinson[125]). In the specimens observed in this series, the overhanging margin was seen to predominate. The lumen of the crater is usually filled with mucus and débris. Beneath this is a rather typical layering or stratification. From within outwards there is first seen a level of necrotic granulation tissue, then a zone of healthy granulations with numerous young capillaries, and finally dense scar tissue. The latter varies greatly in amount and in extent, with the resultant deformity in the adjacent tissues showing marked differences. In general, some distortion is present in all cases showing scarring, and scarring is noted in the greater proportion of all ulcers, except in the acute type. Disturbance in structure due to fibrosis (Table III) was more marked in the gastric group than in the duodenal, but the differences were not significant. Severe degrees of distortion are exemplified by the permanent hour-glass contraction of the stomach and by the shortening which may occur in the pyloropapillary distance in the duodenum. The normal for this distance is given as 8 cm., and it may be reduced to 3 to 4 cm. by contracting fibrous tissue (Robertson in Eusterman and Balfour[52]).

Varying with the age and the activity of the process, the cellular reaction may be of different types and degree. Polymorphonuclear neutrophils are seen in the earlier, more active lesions, while lymphocytes and plasma cells

predominate in those of longer duration (Boyd[21]). Vascular changes of the nature of peri-arteritis and endarteritis have been reported, and thrombosed vessels may be seen (Boyd,[21] Dible[44]).

The mucosa about the crater is often distorted by scar tissue and bizarre formations may result due to regeneration and proliferation. The down-growing epithelium may be pinched off by fibrocytes, leaving packets of epithelium to be seen in the section. The muscularis mucosa may be fragmented or hypertrophied (Steinberg[141]). Edema, to a greater or lesser degree, is apparent in the adjacent submucosa.

Complications and Mortality.—A discussion of the mortality in peptic ulcer is essentially that of the complications of the disease. Patients do not die of peptic ulcer *per se*, but of hemorrhage, perforation, carcinomatous change, obstructive phenomena or the operative procedures undertaken to correct these or to relieve severe symptoms.

Hemorrhage is so common that it may be regarded as a symptom, according to Karsner.[82] It occurs when an ulcer, acute or chronic, involves a vessel as the necrotizing process extends (Boyd,[21] Karsner[82]). Bleeding may be slight or massive, and the latter is sometimes fatal. Protective thrombi may be dislodged by peristalsis, distention or an increase in systemic blood pressure (Karsner[82]). Hemorrhage, according to Barclay,[12] is unrelated to the ulcer's size, site, or even presence. Others, including Goldman[64] and Boyd,[21] consider bleeding to be more frequent in posterior wall duodenal ulcers and lesser curvature gastric ulcers. The gastroduodenal artery and its source, the superior pancreaticoduodenal artery, are involved in duodenal ulcers, and the coronary (gastric) arteries in gastric ulcers (Goldman[64]). The involved arteries may be held open as a result of the stiffening of their walls by fibrous tissue (Robertson in Eusterman and Balfour[52]). Rivers in Eusterman and Balfour[52] expresses the view that duodenal ulcer is the most frequent of all causes of gastro-intestinal bleeding, and that 90.5 per cent of such ulcers bleed at some time. Table IV shows melena and hematemesis to be equally prevalent in this series, and that gastric and duodenal ulcer patients suffer these complications in about equal proportions although somewhat less perhaps than those with combined lesions. In general, slightly more than one-third of the patients bled to some degree, a figure that is in accord with that of several others (Balfour,[11] Emery and Monroe,[48] Goldman,[64] Hurst and Ryle,[75] Osler,[111] MacGuire,[95] Lynch,[94] and Paterson[113]). Smithies[138] recorded a frequency of 36.4 per cent of hemorrhage, with hematemesis occurring in 81 per cent of these and melena in the remaining 19 per cent. This predominance of hematemesis over melena is in contrast to the findings of Chace[28] and of Brown in Cecil,[27] who express the opinion that melena occurs in about 25 per cent of ulcer patients and hematemesis in less than 10 per cent. Gaither[59] and Eggleston,[47] as well as Hurst and Stewart,[76] place the frequency at 18 to 19 per cent. As noted above, there was no difference between the percentages of bleeding ulcers in the gastric or duodenal groups in this series. Some authors have not found this to be true, notably

Blackford and Dwyer,[17] who found massive hemorrhage to be twice as frequent in the stomach (30 per cent) as in the duodenum (15 per cent). Crohn[36] is in agreement with the increased tendency of gastric ulcers to bleed in comparison with those in the duodenum, as are de la Viesca,[42] Percy and Beilin,[115] and Eusterman and Balfour.[52] Roof,[127] however, feels that duodenal ulcers bleed more frequently than gastric.

Again it is here apparent that a difference in the source of the cases studied may account for the variation in the impressions gained by the writer. For example, Hurst and Stewart[76] agree with the statistics of Crohn[36] who places the frequency of hemorrhage at 20 per cent for hospital cases and 10 per cent for all cases of peptic ulcer. This seems to be a fairly reasonable estimate. It is of interest that Somervell and Orr,[139] in their group of cases in India, found hemorrhage exceedingly rare.

Perforation (Table IV) occurred with equal frequency in gastric and duodenal ulcers. Goldman,[64] in a large number of cases, obtained a percentage exactly in accord with the finding in the series herewith reported; namely, 23 per cent. This figure is somewhat higher than that of Miller, Pendergrass and Andrews,[104] who found 15 per cent in their series of operated cases. Roof[127] found that ruptured ulcers made up 18 per cent of his gastric and 20.4 per cent of his duodenal cases. Among other investigators the usual figures are around 5 to 10 per cent (Blackford and Dwyer,[17] DeLario,[41] Emery and Monroe,[48] Lynch[94]). These figures vary in different localities, as do so many statistics. Somervell and Orr,[139] from India, report only four perforations in 2,500 operatively proven ulcer patients.

The average age in patients with perforated ulcers is somewhat lower than that for the group as a whole. Judin,[80] Sallick,[129] and Miller, Pendergrass and Andrews[104] are in agreement as to this fact. Perforation is more frequent in ulcers of short duration, often with histories of no more than a few days or weeks (Boyd[21]). This was most striking in the present series, in which a surprising number had an acute episode, associated with rupture and resultant peritoneal irritation, as the presenting symptom of ulcer. Emery and Monroe,[48] among others, have commented upon this dramatic onset. Patients with histories suggesting the presence of fibrotic, chronic ulcers seldom perforate with symptoms of peritonitis. More often penetration gradually occurs into the adjacent viscera, particularly the liver and pancreas (Boyd[21]).

The controversial subject of the origin of carcinoma in previously benign gastric ulcers is met with frequently in the extensive literature on peptic ulcer. Using the criteria of Chang[29] and others, which criteria include a history of sufficiently long duration (at least two years, and a previous roentgenologic diagnosis of ulcer if possible), location of the lesion in the region of greatest frequency for benign ulcers, and the presence of malignant changes only on one margin and not in the base of the crater, a percentage of 8 was found in gastric ulcers (Table III). This figure is admittedly open to question, as no two observers might interpret the same material in the same

manner (Scott,[132, 133] Spilsbury[140]). It is the belief of Hinton and Trabek[69] that carcinoma never arises in benign ulceration and Morley[105] considers it exceedingly rare. Sauer[130] felt that such a process occurred in but one of 182 gastric ulcer cases. The most frequent figure appearing in the literature on the subject is about 5 to 6 per cent or less (Balfour,[10] Boyd,[21] Cabot and Adie,[23] Brown in Cecil,[27] Chang,[29] Dible,[44] Emery and Monroe[48] Ewing,[53] Hurst and Stewart[76]). Osler[111] indicates a 5 to 10 per cent possibility, and Stewart[143] found evidence of carcinoma in 14 of 216 cases at autopsy, or at operation. Roof[127] and Walton[151] gave 10 per cent as the incidence, while Eusterman and Balfour[52] found malignant changes in 10 to 15 per cent of resected gastric ulcers. Thus it is apparent that there is no definite agreement, and that the problem is not an easy one either clinically or pathologically. Bloomfield[19] divides gastric carcinomata into two main groups—those which arise in stomachs showing chronic gastritis and anacidity, and those with ulcer. Landon[90] is convinced that carcinoma is a sufficiently frequent follower of gastric ulcer to cause concern.

It will be noted in Table II that one case of carcinoma following duodenal ulcer is recorded. In this instance, a long standing, callous ulcer of the duodenum was found in contact with a carcinoma of the stomach, one lying on either side of the pyloric ring. The association, of course, cannot be proven. Carcinomata originating in duodenal ulcers must be exceedingly rare, as is malignant disease of the duodenum. Jefferson,[78] in analyzing all of the reported cases of duodenal carcinoma, found only 31 apparently having their origin in preexisting duodenal ulcer.

The mortality and causes of death found in this study are shown in Table IV. These figures cannot be considered as a fair statistical average, for being hospital cases they represent the relatively more severe. Death due to ulcer was highest in the combined and gastric groups, being almost twice as frequent as in the duodenal. Perforation accounted for the most deaths in the combined ulcer group, carcinoma in the gastric and operative procedures in the duodenal. The latter figure is to be anticipated when the large number of operative cases reported in this series is considered. The total operative mortality is, however, extremely low in this duodenal group; namely, 8.7 per cent, even though this was most frequently the cause of death. Death occurred in a somewhat later age period than that in which the patients were first seen. Death due to carcinoma arising in ulcer was about at the same average age as that for the ulcer group as a whole.

Goldman[64] found the mortality in a group of 890 patients to be 17 per cent. Of those with perforation 32 per cent died, a figure above that obtained in this series (14 to 16 per cent—not shown on the table). Eleven point one per cent of patients with hemorrhage died in this group, which agrees with the Presbyterian Hospital figures (10 per cent of duodenal and 15 per cent of gastric ulcers). Blackford and Dwyer[17] report a mortality of eight in 207 duodenal ulcer cases, a percentage of 3.9. Of these, two deaths were due to hemorrhage, and three each succumbed following operation and perforation.

DeLario's[41] series of 105 peptic ulcers showed death due to ulcer in only two cases, both of which were postoperative. Emery and Monroe[48] showed a death rate of 7.2 per cent, and of those cases with perforation, 28 per cent ended fatally. Hunt,[74] in a recent report, estimates that the mortality in peptic ulcer is somewhere around 2 to 5 per cent, which, in view of the marked variation in the statistics of other authors, would seem to be a fair average. Postoperative deaths are probably more frequent than deaths due to other causes associated with ulcers, but such a statement should be qualified by calling attention again to the fact that surgical cases are those which have done poorly under conservative treatment or those which are suffering from severe complications.

Before leaving the subject of the complications of peptic ulcer, mention should be made of the disturbed or altered physiology of the involved viscus. Spasm, deformity, delay in emptying, variations in peristalsis, changes in tonus and hypersecretion are all demonstrable roentgenologically. A discussion of these phenomena, therefore, will be considered in that section.

Much has been written about the relationship of ulcers to disease of the gallbladder and appendix. This association was studied only in the celiotomy group (Table IV). It is difficult to believe that this relationship is significant as here found. A history of an appendicectomy is not always evidence of appendicitis, nor is one of "gallbladder trouble" a diagnostic certainty. Sufficient information was not found on the charts to make this a reliable estimate. A causal relationship between the appendix and peptic ulcer has been previously mentioned. Aaron[1] was impressed by the physiologic relationship of these structures and noted that pressure on the appendix caused pyloric spasm and epigastric pain. In groups where this problem has been carefully analyzed the frequency of an association is often recorded as higher. Alvarez[7] found gallbladder disease in 8.5 per cent of male gastric ulcer patients and in 13.3 per cent of female patients. In the duodenal ulcer patients 11 per cent of the men and 27 per cent of the women had associated disease of the gallbladder. Rivers and Mason[123] found 13 per cent of their operated duodenal ulcers to have definite cholecystitis pathologically, and in another 3.3 per cent the findings were suggestive. Their gastric cases showed 7.8 per cent of probable gallbladder disease. Emery and Monroe[48] reported associated gallbladder disease in 13 per cent of their ulcer cases, and Smithies[138] 14 per cent. In 18 cases of gastric ulcer at postmortem, Cleland[32] found gallstones in three. From the other side the statistics are less impressive. Hartman and Rivers[65] found ulcer in 3.2 per cent of 879 cases of cholecystitis, which is not much above the usual frequency of the disease as an entity. Laird's[89] percentage of 4.1 led him to believe that the relationship was not direct but slight. As a whole the recorded findings suggest that this is true.

A possible relationship between appendiceal disease and peptic ulcer has not been proved (Emery and Monroe[48]). Hartman and Rivers[65] reported chronic appendicitis to be present in 35.7 per cent of gastric and 44.4 per cent of duodenal ulcer patients. Smithies'[138] rate was 36 per cent and Lari-

TABLE IV

ANALYSIS OF COMPLICATIONS AND MORTALITY

	Medical Pathology			Surgical Pathology			Celiotomy			Totals		
	G.U.	D.U.	Comb.	G.U.	D.U.	Comb.	G.U.	D.U.	Comb.	G.U.	D.U.	Comb.
Complications												
Hemorrhage												
Hematemesis	13 (39%)‡	20 (49%)‡	4 (36%)‡	17 (39%)‡	19 (33%)‡	4 (36%)‡	10 (27%)‡	55 (31%)‡	5 (38.5%)‡	40 (35%)‡	94 (34%)‡	13 (37%)
Melena	18 (55%)	30 (75%)	6 (54%)	16 (36%)	26 (46%)	5 (45%)	7 (19%)	50 (29%)	5 (38.5%)	41 (36%)	106 (39%)	16 (46%)
Perforation	11 (15%)§	21 (26%)§		5 (9%)§	10 (16%)§		22 (43%)§	30 (22%)§		38 (24%)§	70 (22%)§	
Av. age at perforation	49 yrs.	48.2 yrs.		39.4 yrs.	36.3 yrs.		35.3 yrs.	37.6 yrs.		39.8 yrs.	40.5 yrs.	
Operations												
No. of cases operated upon	18 (24%)§	24 (30%)§	9 (47%)§	44 (100%)§	60 (100%)§	11 (100%)§	38 (100%)§	180 (100%)§	13 (100%)§	100 (63%)§	264 (83%)§	33 (77%)§
History of appendicectomy							5 (12%)§	11 (9%)§				
History of gallbladder disease							1 (3%)§	12 (6%)§				
Mortality												
Death due to ulcer	25 (34%)*	27 (35%)*	10 (5%)*	9 (20%)*	4 (7%)*	2 (18%)*	8 (21%)*	9 (5%)*	1 (8%)*	42 (27%)*	40 (13%)*	13 (30%)*
Hemorrhage	5 (20%)†	8 (30%)†	0 (0%)†	0 (0%)†	0 (0%)†	0 (0%)†	0 (0%)†	0 (0%)†	0 (0%)†	5 (12%)†	8 (20%)†	0 (0%)†
Perforation	3 (12%)	6 (22%)	5 (50%)	0 (0%)	0 (0%)	2 (100%)	3 (37%)	3 (33%)	0 (0%)	6 (14%)	9 (23%)	7 (54%)
Postoperative	8 (32%)	13 (48%)	5 (50%)	5 (55%)	4 (100%)	0 (0%)	2 (26%)	6 (67%)	1 (100%)	15 (36%)	23 (57%)	6 (46%)
Carcinoma	9 (36%)	0 (0%)	0 (0%)	4 (45%)	0 (0%)	0 (0%)	3 (37%)	0 (0%)	0 (0%)	16 (38%)	0 (0%)	0 (0%)
Av. age with death due to ulcer	53.2 yrs.	52.3 yrs.		56.4 yrs.	52.2 yrs.		54 yrs.	49.9 yrs.		54.3 yrs.	51.7 yrs.	
Av. age with death due to carcinoma in old ulcer	53 yrs.			61 yrs.	57 yrs.		56 yrs.			55.5 yrs.	57 yrs.	

* Percentage of all ulcers.
† Percentage of cases with death due to ulcer.
‡ Percentage of patients having complete records (Table VI).
§ Percentage of ulcer patients.

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more[91] noted that 18 per cent of his cases had had appendicectomies. Somervell and Orr,[139] in statistics from India, found a 73 per cent concomitance.

Symptomatology.—That the symptomatology and history of peptic ulcer are characteristically those of periodicity, persistence and variable regularity, is almost axiomatic (Osler,[111] Brown in Cecil,[27] Hunt,[74] Emery and Monroe,[48] *etc.*). Ulcer patients usually have long histories, Hunt,[74] noting that it was usually two years before the patients presented themselves to a physician. The average duration of symptoms in Emery and Monroe's series[48] was seven years, and Roof's[127] was 6.5 years for duodenal and 5.5 years for gastric ulcer patients. More than half of Smithies'[138] cases, and there were 500 gastric ulcers, had had symptoms for from 5 to 20 years. Chang[29] placed 33 per cent of his gastric cases in this category. The average duration of symptoms in our own series was 7.8 years for gastric and 7.3 years for duodenal ulcers. This is closely in line with the above findings (Table V).

The periodicity of symptoms is emphasized by the ratios noted in Table V. They were intermittent rather than constant, six times as frequently in the gastric and 24 times as frequently in the duodenal cases. Remissions occurred in 61 per cent of gastric ulcer patients and 64 per cent of duodenal ulcers in the summary by Miller, Pendergrass and Andrews.[104] Smithies[138] found this phenomenon characterizing 69 percent of his cases, and Roof[127] commented on the history of "attacks" in 81.2 per cent of duodenal and 78.5 per cent of gastric ulcers. The occurrence of exacerbations in the spring and fall has been remarked frequently (DeLario[41]). It was Smithies'[137] belief that the explanation was to be found in the prevalence of "epidemic infectious agents" at these times. Eusterman and Balfour[52] found this seasonal variation in 50 per cent of their series. They offered no satisfactory explanation but suggested, in agreement with Smithies, that there might be an increased infectious activity, or possibly that some nutritional factor entered into the question at these times. Hurst and Stewart[76] state that the return of symptoms may "definitely date from a sudden change in the weather"; "The effect of weather is not entirely direct; it depends in part on the increased liability to nasal catarrh, tonsillitis, bronchitis, and 'influenzal attacks' in bad weather." It is their belief that this seasonal change is much less marked in gastric than in duodenal ulcer patients.

Pain is the most constant and important symptom of peptic ulcer. The frequency and characterization of the pain in the present series of cases is noted in Table V. The descriptive adjectives used are those employed by Miller, Pendergrass and Andrews.[104] The percentages of 72, 88 and 83 respectively for gastric, duodenal and combined ulcer patients indicate the very general occurrence of this symptom. Hinton[70] recorded it in almost the exact proportion, 79 per cent. Other illustrative statistics include those of Lynch,[94] with pain present in 69 per cent of gastric and 73 per cent of duodenal cases; Percy and Beilin,[115] with 100 per cent of gastric and 92.4 per cent of duodenal cases; and Smithies[138] who noted pain in 98 per cent of his gastric ulcers.

TABLE V

ANALYSIS OF SYMPTOMATOLOGY

	Medical Pathology			Surgical Pathology			Celiotomy			Totals		
	G.U.	D.U.	Comb.	G.U.	D.U.	Comb.	G.U.	D.U.	Comb.	G.U.	D.U.	Comb.
Symptoms												
Average duration	8.5 yrs.	7.3 yrs.		8.3 yrs.	7.9 yrs.		6.3 yrs.	7.0 yrs.		7.8 yrs.	7.3 yrs.	
Intermittent	14	25	5	37	52	10	29	164	10	80	241	25
Constant	2	1	0	6	2	1	6	7	2	14	10	3
Ratio	7:1	25:1	5:0	6:1	26:1	10:1	5:1	23:1	5:1	6:1	24:1	8:1
Pain												
Present	33 (45%)*	37 (52%)*	11 (61%)*	41 (95%)*	56 (95%)*	11 (100%)*	36 (97%)*	175 (98%)*	12 (100%)*	110 (72%)*	264 (88%)*	34 (83%)*
Vague or absent	40 (55%)‡	34 (48%)	7 (39%)	2 (5%)	3 (5%)	0 (0%)	1 (3%)	4 (2%)	0 (0%)	43 (28%)	41 (12%)	7 (17%)
Type												
Severe	4 (12%)†	10 (27%)†	1 (9%)†	20 (49%)†	22 (40%)†	5 (45%)†	17 (47%)†	37 (21%)†	2 (17%)†	41 (37%)†	69 (26%)†	8 (24%)†
Dull	4 (12%)	6 (16%)	5 (45%)	6 (15%)	13 (23%)	1 (9%)	5 (14%)	48 (27%)	4 (34%)	15 (14%)	67 (25%)	10 (29%)
Sharp	2 (6%)	3 (8%)	1 (9%)	5 (12%)	7 (13%)	3 (27%)	3 (9%)	32 (18%)	1 (8%)	10 (9%)	42 (16%)	5 (15%)
Gnawing	5 (15%)	6 (16%)	5 (45%)	4 (10%)	10 (18%)	0 (0%)	7 (19%)	22 (13%)	1 (8%)	16 (15%)	38 (14%)	6 (17%)
Cutting	3 (9%)	3 (8%)	0 (0%)	5 (12%)	2 (4%)	1 (9%)	2 (6%)	5 (3%)	1 (8%)	10 (9%)	10 (4%)	2 (6%)
Burning	5 (15%)	1 (3%)	2 (18%)	6 (15%)	11 (20%)	3 (27%)	10 (22%)	26 (15%)	2 (17%)	21 (19%)	38 (14%)	7 (21%)
Aching	0 (0%)	0 (0%)	0 (0%)	2 (5%)	2 (4%)	0 (0%)	0 (0%)	6 (3%)	0 (0%)	2 (2%)	8 (3%)	0 (0%)
Colicky	3 (9%)	8 (21%)	0 (0%)	10 (25%)	4 (8%)	1 (9%)	3 (9%)	29 (17%)	1 (8%)	16 (15%)	41 (15%)	2 (6%)
Fulness or heaviness	6 (18%)	6 (16%)	2 (18%)	14 (34%)	18 (32%)	2 (18%)	13 (36%)	70 (40%)	5 (42%)	33 (30%)	94 (36%)	9 (26%)
Not stated	2 (6%)	8 (21%)	1 (9%)	1 (2%)	0 (0%)	0 (0%)	1 (3%)	1 (0.5%)	1 (8%)	4 (4%)	9 (3%)	2 (6%)
Location												
Epigastric	33 (100%)†	30 (81%)†	10 (90%)†	41 (100%)†	51 (91%)†	11 (100%)†	33 (92%)†	165 (94%)†	11 (92%)†	107 (98%)†	246 (93%)†	32 (94%)†
Rt. hypochond	2 (6%)	4 (11%)	3 (27%)	3 (7%)	6 (11%)	2 (18%)	2 (6%)	8 (5%)	0 (0%)	7 (6%)	18 (7%)	5 (15%)
Lt. hypochond	3 (9%)	0 (0%)	0 (0%)	6 (15%)	3 (5%)	0 (0%)	2 (6%)	3 (2%)	0 (0%)	11 (10%)	6 (2%)	0 (0%)
Lwr. abdomen	2 (6%)	4 (11%)	0 (0%)	1 (2%)	1 (2%)	0 (0%)	4 (11%)	8 (5%)	1 (8%)	7 (6%)	13 (5%)	1 (3%)
Ref. to back	5 (15%)	8 (21%)	3 (27%)	11 (27%)	4 (8%)	3 (27%)	0 (0%)	20 (11%)	0 (0%)	16 (15%)	32 (12%)	6 (17%)
Ref. to shoulder	0 (0%)	1 (3%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	1 (0.5%)	0 (0%)
Chest	0 (0%)	1 (3%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	2 (6%)	1 (0.5%)	0 (0%)	2 (2%)	2 (1%)	0 (0%)
Relation to meals												
None	3 (9%)†	7 (19%)†	4 (36%)†	2 (5%)†	13 (23%) †	3 (27%)†	6 (16%)†	24 (14%)†	4 (34%)†	11 (10%)†	44 (17%)†	11 (32%)†
2 hrs. or more after	11 (33%)	15 (40%)	2 (18%)	14 (34%)	18 (32%)	2 (18%)	5 (14%)	81 (46%)	2 (17%)	30 (27%)	114 (43%)	6 (17%)
1.5–2 hrs.	2 (6%)	0 (0%)	1 (9%)	2 (5%)	3 (5%)	1 (9%)	6 (16%)	13 (7%)	5 (42%)	10 (9%)	16 (6%)	7 (21%)
1–1.5 hrs.	3 (9%)	3 (8%)	1 (9%)	6 (15%)	9 (16%)	2 (18%)	4 (11%)	6 (3%)	0 (0%)	13 (12%)	18 (7%)	3 (9%)
.5–1 hr.	6 (18%)	2 (5%)	2 (18%)	13 (32%)	6 (11%)	2 (18%)	11 (28%)	16 (9%)	0 (0%)	30 (27%)	24 (9%)	4 (12%)
"After"	2 (6%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	1 (9%)	1 (3%)	12 (7%)	0 (0%)	3 (3%)	12 (4%)	1 (3%)
"Before"	1 (3%)	3 (8%)	0 (0%)	2 (5%)	3 (5%)	0 (0%)	1 (3%)	10 (5%)	0 (0%)	4 (4%)	16 (6%)	0 (0%)

* Percentage of all cases.

† Percentage of patients having pain.

‡ Many acute, without symptoms.

The type of pain, however, does not seem to denote the site of the lesion. Severe pain was somewhat more frequent in gastric ulcers. "Dull" and "sharp" were both employed more in the duodenal group. "Cutting" and "burning" were a little more usual in gastric than duodenal ulcers. In the combined group "dull" and "burning" were more frequently employed than in other types of ulcers. The general facts in this descriptive analysis agree with those of Miller, Pendergrass and Andrews.[104] "Sharp, burning" pain was more frequent in their series. Only a small percentage was noted by them to complain of fulness or heaviness, while it will be noted in Table V that approximately one-third of the cases reported such distress. Other reviewers have recorded about the same variations in the intensity of the discomfort (Percy and Beilin,[115] Roof[127]). Emery and Monroe[48] described slight distress in 20 per cent of their cases, moderate in 58 per cent and intense in 18.4 per cent. Of their 539 patients with abdominal distress, 136 applied such adjectives as "boring," "grinding," "gnawing," *etc.*, and about one-half complained of fulness.

The allocation of pain to a portion of the abdomen is noted in Table V. It is seen to be predominantly epigastric, as is generally recognized. Four hundred sixty-one of Emery and Monroe's[48] 556 cases were so located. Percy and Beilin,[115] Chace,[28] Miller, Pendergrass and Andrews,[104] Smithies,[138] Hunt,[74] and Roof[127] have reported this same finding. As might be expected, pain in the right and left hypochondria was more frequent in duodenal and gastric ulcers respectively. Referred pain to the back was present in 15 per cent, with no particular difference in the three types of ulcer. The findings of Miller, Pendergrass and Andrews[104] were similar and somewhat higher than those of Roof,[127] who found this phenomenon more frequent in gastric ulcers, attributing its occurrence to posterior adhesions. Hunt[74] ascribes it, especially, to the presence of adhesions to the pancreas, with pain referred to the cardiac and splenic areas as being associated with lesions higher on the lesser curvature. In this connection the analysis of Rivers[120] is of interest. He reported that pain is poorly localized in 90 per cent of shallow gastric lesions, but definitely placed in 50 per cent of large and 90 per cent of perforating ulcers. In the latter type, where the ulcer involved the mesentery or adjacent viscera, 93 per cent had pain referred to the back or thorax. In duodenal ulcer he found diffuse distress an accompaniment of obstructing lesions. In this region 64 per cent of cases, where the ulcer was not perforating, gave a poorly defined pain distribution, while accurately localizable pain was noted in extensive and subacute ulceration with considerable regularity. In 90 per cent of perforating duodenal ulcers the pain was in the right upper quadrant, and 77 per cent of these remarked that it was also referred to the liver and the back.

The relationship of pain to the time of eating is variable, but it is apparent, from Table V, that pain occurs somewhat earlier in gastric than in duodenal ulcer, and that there is usually some definite relationship. That there is an association is the impression of Miller, Pendergrass and Andrews,[104]

Smithies,[138] Chace,[28] and Emery and Monroe[48] and others. Hurst and Stewart,[76] Roof[127] and Eusterman and Balfour,[52] among others, noted that pain was present earlier after eating in gastric than in duodenal ulcers. Eusterman and Balfour,[52] Smithies,[138] and Hurst and Stewart[76] state that pain occurred earlier in gastric ulcers situated in the cardia than in those farther down in the stomach, appearing as early as one-half hour after eating. Hunt[74] suggested that the nearer the pylorus the ulcer is, the longer the interval between eating and the onset of pain. The regularity of the occurrence of pain, emphasized by Moynihan,[108] did not seem to be frequent or accurate enough to be considered characteristic. Rivers[120] makes the interesting observation that a change in the characteristics of ulcer pain, with increased persistence, despite the use of alkali, denotes a change in the pathology.

The cause of pain in peptic ulcers has not been definitely determined. An increased acidity with resultant irritation of bared nerve fibrils has been suggested, and Palmer[112] produced pain in 95 per cent of tested cases of ulcer by its application. Against this as a universal explanation, is the low acidity in many patients with severe pain (DeLario[41]). Even the relief by alkali might be explained by a resultant decrease in muscle spasm, rather than acid neutralization. Spasm, as a cause of pain, has many adherents. Deaver and Burden[40] and Hurst[77] considered pyloric spasm as the cause of pain. Barclay[12] described the concurrence of pain, spasm and tension as seen fluoroscopically. DeLario[41] believes that pain occurs only when the base of the ulcer is attached to the muscle of the submucosa or to the muscular coat proper, as by edema, adhesions or inflammatory changes. By such fixation, muscular activity results in tension on the ulcer crater and pain. Brown in Cecil[27] agrees with the theory that spasm of the pylorus causes ulcer pain, but adds that neuralgia or neuritis of the gastric nerves may occasionally be responsible. It should be added that many definite ulcers are seen roentgenologically and by direct observation in association with which there has been no pain or discomfort (Boyd[21]).

Table VI shows the number of instances in which case records were adequate for an analysis of other symptoms. Nausea and vomiting were more often encountered in the gastric and combined ulcer groups than in the duodenal, while belching or eructations appear in a larger percentage of the latter. As Miller, Pendergrass and Andrews[104] state, these symptoms are not typical of the disease. The more important symptom, bleeding, has been discussed elsewhere. Constipation is rather frequent, suggesting a close functional relationship between the portions of the gastro-intestinal tract. Anorexia is variable and probably accounts for weight loss, along with food restrictions, vomiting, night pain and lack of sleep (Hurst and Stewart,[76] Smithies[138]).

Vomiting is associated with pain or with pyloric obstruction, either organic or reflex (Chace,[28] Emery and Monroe[48]). As will be discussed later, pyloric obstruction is more frequent in association with gastric ulcers, and

TABLE VI

FURTHER ANALYSIS OF SYMPTOMATOLOGY IN CASES WITH MORE COMPLETE DATA

	Medical Pathology			Surgical Pathology			Celiotomy			Totals		
	G.U.	D.U.	Comb.	G.U.	D.U.	Comb.	G.U.	D.U.	Comb.	G.U.	D.U.	Comb.
Symptoms												
Nausea	9 (27%)*	11 (27%)	5 (45%)	21 (48%)	39 (69%)	7 (63%)	23 (62%)	83 (47%)	5 (39%)	37 (32%)	133 (49%)	17 (50%)
Vomiting	23 (70%)	34 (83%)	10 (91%)	28 (64%)	39 (69%)	11 (100%)	28 (76%)	123 (70%)	11 (85%)	79 (69%)	196 (72%)	32 (92%)
Eructations	19 (58%)	14 (34%)	6 (54%)	34 (77%)	28 (49%)	6 (54%)	17 (46%)	93 (53%)	4 (31%)	70 (61%)	135 (50%)	16 (46%)
Constipation	16 (48%)	14 (34%)	6 (54%)	25 (57%)	24 (42%)	5 (45%)	15 (41%)	83 (47%)	6 (45%)	56 (49%)	121 (44%)	17 (50%)
Diarrhea	5 (15%)	2 (5%)	1 (9%)	1 (2%)	2 (4%)	0 (0%)	1 (3%)	0 (0%)	0 (0%)	7 (6%)	4 (1%)	1 (3%)
Anorexia	4 (12%)	9 (22%)	3 (27%)	9 (21%)	9 (16%)	1 (9%)	5 (13%)	23 (29%)	2 (15%)	18 (16%)	51 (19%)	6 (17%)
Weight loss	20 (61%)	18 (44%)	9 (81%)	30 (68%)	27 (47%)	4 (36%)	18 (32%)	94 (53%)	8 (61%)	68 (60%)	139 (51%)	21 (60%)
Av. loss	20	25	18.75	20	19	18.5	18	15.5	22	20	17	20
Pounds lost per month	2.25	1.25	1.5	1.5	2.5	4	1.3	2.25	1.0	1.7	2.0	2.0
Cases complete enough for study*	33	41	11	44	57	11	37	175	13	114	273	35
Physical Findings												
Local tenderness	16 (50%)	17 (41%)	9 (81%)	29 (66%)	32 (56%)	9 (81%)	31 (84%)	109 (62%)	5 (39%)	76 (67%)	158 (58%)	23 (66%)
Rigidity	2 (6%)	8 (20%)	3 (27%)	4 (9%)	5 (9%)	3 (27%)	15 (41%)	27 (15%)	1 (8%)	21 (18%)	40 (15%)	7 (20%)
Spasm	2 (6%)	9 (22%)	0 (0%)	4 (9%)	5 (9%)	1 (9%)	9 (24%)	33 (19%)	0 (0%)	15 (14%)	47 (17%)	1 (3%)
Visible peristalsis	4 (12%)	2 (5%)	1 (9%)	1 (2%)	2 (4%)	0 (0%)	2 (5%)	9 (5%)	1 (8%)	7 (6%)	13 (5%)	2 (6%)
Relief												
Alkali	18 (55%)	24 (59%)	7 (63%)	29 (66%)	34 (60%)	6 (55%)	25 (68%)	134 (77%)	10 (77%)	72 (63%)	192 (70%)	23 (66%)
Vomiting	9 (27%)	5 (12%)	0 (0%)	12 (27%)	17 (30%)	1 (9%)	4 (11%)	51 (29%)	1 (8%)	25 (22%)	73 (27%)	2 (6%)
Food	11 (33%)	15 (38%)	3 (27%)	17 (39%)	21 (37%)	4 (36%)	5 (13%)	97 (55%)	5 (39%)	33 (29%)	133 (49%)	12 (35%)
Pressure	0 (0%)	0 (0%)	0 (0%)	3 (7%)	1 (2%)	0 (0%)	0 (0%)	1 (1%)	0 (0%)	3 (3%)	2 (1%)	0 (0%)
Eructations	0 (0%)	1 (3%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	1 (3%)	18 (10%)	0 (0%)	1 (1%)	19 (7%)	0 (0%)

* All percentages are those computed upon the basis of the number of cases judged adequate in clinical data for analysis of other symptoms.

may account for the increased frequency of vomiting in cases with such ulcers. Although vomiting was not much more frequently met with in gastric cases in this series, it was in those of Eusterman and Balfour,[52] Hunt,[74] Jordan,[79] Lynch[94] and Roof.[127]

Physical Findings.—Definite physical findings are few. The principal ones are noted in Table VI. Of these, local tenderness was the most consistently found, and this more often in gastric than in duodenal ulcers. Rigidity and spasm were not unusual, while a palpable mass and visible peristalsis were comparatively rare. The findings of Miller, Pendergrass and Andrews[104] closely agree, and this is the usual impression. Hurst and Stewart[76] stress the diagnostic value of the finding of a localized area of tenderness or hyperesthesia. They found less rigidity and spasm in the absence of pain. The finding of a "tumor" was rather unusual in their experience.

Relief.—As is well known, alkali and food, as well as vomiting, may give relief in ulcer patients, and are more effective in duodenal than in gastric ulcers. This is also noted in the reports of Miller, Pendergrass and Andrews[104] and of Roof.[127] Other sources of relief, mentioned infrequently, were pressure, catharsis, changes of posture and eructations.

Roentgenologic Phenomena.—Moynihan[108] states: "Of all ancillary methods of diagnosis that of the radiologist should be of the greatest value. In the diagnosis of gastric ulcer it has pride of place; in competent hands it is far more accurate than any other method of diagnosis, clinical or chemical, or than all other methods combined." Not only is the presence of an ulcer to be determined, but the roentgenologist has the opportunity to evaluate the severity of the complications, and the efficacy of treatment. The roentgenologic diagnosis of peptic ulcer depends upon the demonstration of "direct and indirect" signs. The former is the crater, the latter are those due to disturbances in outline or function of the stomach or duodenum.

The pocket formed by a crater is, of all evidence of ulcer, the most definite and the only positive proof (Barclay,[12] Carman,[25] Golden[63]). Its first description is variously ascribed to Reiche[119] and to Haudek.[67] It would seem that Reiche, in 1909, demonstrated the niche in gastric ulcers, and Haudek, in 1912, in duodenal ulcers.

If the gastric ulcer is superficial, involving only the mucosa, the crater may be seen in profile as a small projection beyond the gastric outline, or merely as an opaque spot if on the anterior or posterior surfaces. Such a finding is best obtained with a small amount of opaque medium in the stomach. Radiating mucosal folds may be seen centering about such an area, a finding emphasized by Forssell.[54] If there is a greater penetration, the niche may be seen as a projection beyond the gastric contour. Its depth is relatively great as compared to its diameter in the majority of instances (Golden[63]). The depth of a crater shadow may be increased by a heaping up of mucosa around the mouth due to edema, spasm of the muscularis mucosa or scar tissue (Barclay[12]). Such a penetrating ulcer is usually not fixed to the adjacent structures (Hurst and Stewart[76]).

When the ulcer penetrates beyond the gastric musculature, an accessory pocket occurs (Kirklin in Eusterman and Balfour[52]). In such pockets a fluid level with a superimposed gas bubble may be seen (Kirklin in Eusterman and Balfour,[52] Golden,[63] Holmes and Ruggles[71]). If adjacent viscera, most frequently the pancreas and the liver, are invaded by this process, the area may be fixed to palpation, but if it is the gastrohepatic omentum this fixation does not occur (Kirklin in Eusterman and Balfour,[52] Hurst and Stewart[76]). When the liver is the site of such imbedding, the crater may be seen to descend with respiration, a finding not noted in those instances where the pancreas is involved (Kirklin in Eusterman and Balfour[52]). Failure to visualize these craters may be due to foreign material which acts as a plug, as in a case reported by Stewart and Illick.[144] Such foreign material and haste in examining are considered by them to be the causes, most frequently, of failure to visualize gastric ulcer craters.

Twining[148] has emphasized the rolled edges of the benign ulcer crater in contradistinction to the triangular, more or less sharp outline of malignant excavations. Scott,[132, 133] Rivers and Dry,[122] and Jordan[79] are among the many who have emphasized the difficulty of differentiating the benign from the carcinomatous niche. Scott,[132, 133] and Singleton[135] urge a course of medical treatment and repeated examinations to distinguish between these two conditions, judgment depending upon the presence or absence of healing.

If the crater is on the lesser curvature it is usually readily seen (Hurst and Stewart[76]). If the crater is on the anterior or posterior surfaces of the stomach, or if small, considerable painting of the slightly filled viscus may be required before it is seen (Barclay[12]). Pressure and rotation of the patient in order to view the crater in profile are often necessary (Barclay,[12] Golden[63]). Craters in the region of the cardia may be extremely difficult to demonstrate, palpation often being impossible. A change in position with lowering of the patient's head and a minimal filling may materially aid in its disclosure (Barclay[12]). The niche in an ulcer of the pyloric region is frequently unseen, but variations in the function and appearance of the region should stimulate careful observation (Barclay[12]). Jordan[79] has pointed out that a loop of intestine, particularly the duodenojejunal flexure, may be confused with lesser curvature craters.

The secondary or indirect evidences of gastric ulcer depend upon disturbances in the physiology of the stomach and often upon deformities resulting from spasm or scarring. An incisura or notching opposite a crater is an infrequent finding and thus is of little diagnostic significance (Golden[63]). Moreover, it may be due to reflex stimuli from disease of the gallbladder, appendix or duodenum (Carman[25]). Such an incisura, to be of significant import, must be constant, in the same location, present in all positions assumed during the course of the examination, and withstand manipulation and atropinization (Carman[25]). If the incisura is deep enough, subsequent fibrosis may result in an "hour-glass" or bilocular stomach. The neck, or channel at the site of the ulcer and incisura, may be more or less permanent (Kirklin

in Eusterman and Balfour,[52] Golden[63]). Clairmont[30] distinguished the hour-glass deformity associated with annular carcinoma from that due to benign ulcer, describing the displacement of the greater curvature toward the lesser in the benign lesion, and the lesser toward the greater in the presence of carcinoma. This gives rise to the so-called "B" and "X" types of deformity. Roof,[127] in 1934, recorded an 8 per cent frequency in his gastric ulcer cases. Apparently hour-glass stomachs are now less frequently seen than 10 or 20 years ago (Twining[148]).

Gastrospasm varies greatly in amount and persistence. It may be diffuse or localized, but it is most commonly found in the prepyloric region regardless of the location of the gastric ulcer (Kirklin in Eusterman and Balfour[52]). This may be attributed to an intrinsic reflex or to inflammatory changes in the pyloric region.

Retention after six hours may or may not be noted. It would seem to be more frequently found in association with gastric than with duodenal ulcers (Roof[127]). Carman[25] found it present in 55 per cent of gastric ulcers, and Miller, Pendergrass and Andrews[104] in 60 per cent of all cases. Eusterman[50] noted retention in 29 per cent of gastric ulcers, 35 per cent of which were sufficiently removed from the pylorus to suggest a reflex phenomenon. In his duodenal cases, retention was slightly less frequent, a percentage of 26. Berkman[15] also found retention more frequently in gastric than in duodenal lesions and he too remarked upon the fact that the pylorus was involved in only 22.7 per cent of gastric and less than 50 per cent of duodenal ulcers. Others who report high frequencies of retention in gastric ulcers include Smithies,[138] with a percentage of 67, and Chang,[29] who set it at 50 per cent. In the analysis of Miller, Pendergrass and Andrews,[104] pylorospasm was present in 48 per cent of gastric ulcers but organic obstruction in only 8 per cent. Alvarez, Horowitz and Ascanio,[8] in studies on the pyloric muscle, concluded that gastric ulcers generally caused pyloric hypertrophy, but that duodenal ulcers seldom if ever did unless there was actual organic obstruction. This is an interesting commentary upon the possibility of the reflex irritating effect of an ulcer at some distance from the pylorus.

Retention, it should be added, is not diagnostic, in any sense, of the presence of a gastric lesion. Maingot's[96] synopsis of the conditions leading to gastric retention is interesting in this connection. He divides the causes into general and abdominal. Of the former he lists: (a) Nervous and physiologic (*e.g.,* migraine, melancholia); (b) endogenous toxins (*e.g.,* nephritis, infections); (c) exogenous toxins (*e.g.,* emetics). The abdominal causes may be extraperitoneal, such as renal colic, or intraperitoneal, such as peritonitis, cholecystitis, peptic ulcer, neoplasm or pyloric obstruction.

The tone of the stomach may be lowered, a finding more frequently noted in patients whose stomachs have decompensated due to long standing pyloric obstruction (Carman,[25] Golden[63]).

The changes in peristalsis are never characteristic of gastric ulcer. It may be increased (Holmes and Ruggles[71]), or weak and irregular (Car-

man[25]). It may be interrupted at the site of the lesion if the ulcer involves a sufficient depth of tissue (Golden[63]). Fraenkel[55, 56] described a functional defect in motility which he ascribed to alteration in the activity of the transverse musculature. This he considered due to irritation, and described it as a failure of movement of a small segment of gastric contour about an ulcer—an area of flattening of 8 Mm. or so in length. He called this the "cross-bar sign." It is best seen on serial roentgenograms. Miller, Pendergrass and Andrews[104] found peristalsis to be hyperactive in 59 per cent of their gastric cases, but more often so with duodenal ulcers, 65 per cent, suggesting a reflex, nervous association. They also reported peristalsis as normal in 15 per cent and sluggish in 22 per cent of their gastric cases, normal in the same per cent, 15, of duodenal and sluggish in 15 per cent.

Hypersecretion is sometimes seen as an evidence of irritation or stasis (Golden[63]).

Pyloric ulcers require somewhat more discussion because of the difficulties attendant upon their diagnosis. Barclay[12] bases many obscure diagnoses on secondary phenomena because of the difficulty with which the demonstration of the crater is so often accompanied. Local tenderness, hyperperistalsis, intermittent spasm and asymmetry of the pyloric opening into the bulb, have all helped to suggest the presence of an ulcer. The latter deformity is more often found with duodenal ulcer than with pyloric. It is also described by Kerley.[83] Stone and Ruggles,[145] discussing the various changes to be noted in the prepyloric and pyloric regions, attributed an eccentric pylorus to either an anatomic variation, adhesions, irregularities in the muscularis mucosa, or an adjacent ulcer.

As in the stomach, the demonstration of the barium filled niche is the most certain evidence of ulceration in the duodenum (Åkerlund,[2] Barclay,[12] Berg,[14] Carman,[25] Kirklin in Eusterman and Balfour,[52] Golden,[63] Kerley,[83] Kirklin and Burch,[86] Peter,[116] *etc.*). A niche without deformity is rare unless the ulcer is extremely superficial (Åkerlund[4]). George and Gerber[62] expressed the opinion that any duodenal ulcer will deform the duodenum, that a normal bulb on the roentgenogram, therefore, rules out the possibility of any "surgical ulcer." However, craters have been seen without associated deformity (Golden,[63] Kirklin in Eusterman and Balfour[52]). Palpation and pressure play an important rôle in the visualization of the bulb and the maintaining of the shadow of a crater until a roentgenogram can be taken (Barclay[12]). This is often difficult, and the more modern technic of "pressure films" has been a definite aid. Using such a technic the number of craters visualized has increased remarkably. Åkerlund[2] advocated such pressure methods because of the frequency of *"en face"* ulcer craters, on the anterior or posterior wall, and because of the possibility of multiple lesions being missed in a fully distended, uncompressed bulb. Using such equipment he demonstrated the crater in 60 per cent and 75 per cent of cases as noted in two reports Åkerlund[3, 4]), Berg[14] noted a crater in 50 per cent of duodenal ulcers,

Diamond[43] in 66.6 per cent, Clark and Geyman[31] 54 per cent and Ettinger and Davis[49] in 50 per cent. Golden[63] stated that craters are demonstrable in 50 to 60 per cent of cases with special methods and in 13 to 15 per cent without. Carman[24] used no pressure apparatus and had a frequency of demonstration of a crater in duodenal ulcers of 27 per cent, while Kirklin,[84] and Kirklin and Burch,[86] showed them in 15 and 17 per cent of two series. In the latter report[86] they stated that a crater must retain its position despite heavy palpation to be definitely diagnostic. They felt that the higher frequency of niche demonstration with pressure apparatus was undoubtedly real, but could lead to errors in other directions. For example, such a technic might lead to the diagnosis of a crater when there was present a cicatricial sacculation, pseudodiverticulum or merely a fleck of barium caught between the folds of the mucosa. They also felt that the discrepancy in crater frequencies might be due to the criteria used, or the source of material studied. For example, they found higher percentages in surgical cases, and only 25 per cent of duodenal ulcers (140 cases in all) at necropsy showed definite craters. Kirklin and Burch, however, stress the importance of the niche diagnosis and add this differential suggestion. A niche means an ulcer if deformity is present, an active ulcer if there is also irritability, and an improving ulcer if it grows smaller or disappears. Holmes and Schatzki,[73] it might be added, feel that the distortion of the mucosal relief about a niche is of greater importance and more conclusive evidence of the presence of a crater. A "halo" of swollen mucosa about the crater has been described by Peter,[116] and a heaping up to increase the apparent depth of penetration has been noted here as in the stomach (Barclay[12]).

Of the indirect signs, distortion of the bulbar outline and of its mucosal pattern are so prevalent that they may well be considered direct evidence. These were first described by Cole[33] and ascribed by him to the presence of a duodenal ulcer. Such a deformity of the bulb may be due to other causes, including incomplete filling, periduodenal adhesions secondary to upper, right quadrant inflammatory processes (particularly gallbladder disease), neoplasm, spasm from extrinsic sources, duodenitis and healed ulcer (Kirklin in Eusterman and Balfour,[52] Cordiner and Calthrop[34]). Still, the aggregate of all these causes is small and should seldom lead to an incorrect diagnosis (Kirklin in Eusterman and Balfour[52]). Åkerlund[4] indicated that the confusing irregularities in bulbar outline could usually be sufficiently clarified in order to reach a diagnosis. Thus a rounded shadow due to the impression of the gallbladder can usually be moved on pressure or may be differentiated on rotating the patient. Such a deformity is usually seen in the distal bulb, and can, in the last analysis, be excluded by a gallbladder series employing tetraiodophenolphthalein. Periduodenal adhesions result in a decreased mobility of the bulb, and the deformity is somewhat atypical. New growths are rare, benign lesions such as polypi giving a rounded shadow. Congenital diverticula seldom arise from the bulbar portion of the duodenum, and if projected from the second portion in a confusing manner can often be displaced by manipula-

tion or rotation. Barclay[12] states that a positive diagnosis is usually correct if based on deformity alone, but never certain unless the niche is also seen.

Deformity of the bulb is the result of spasm and scarring (Berg,[14] Köhler,[87] Sutherland[147]). With the spasm limited to the muscularis mucosa, a star formation of radiating lines may result (Berg,[14] Golden,[63] Holmes and Ruggles,[71] Peter[116]). More often the outer musculature is involved to some degree. Opposite the crater an incisura, similar to that found in the stomach, may be seen (Berg,[14] Kirklin in Eusterman and Balfour,[52] Peter[116]). No more than a loss of the normal convexity of the bulb may be noted in the area around the crater (Peter[116]). By far the greater number of duodenal ulcers, however, are associated with a marked, often bizarre outline.

The bulb may be shortened markedly, or narrowed to a thin channel (Åkerlund,[4] Berg,[14] Köhler,[87] Peter[116]). Pseudodiverticula, or pouches with the crater at the stoma, may arise due to contraction of scar tissue bands and the pulsion activity of food (Åkerlund[2]). As previously mentioned, an eccentric position for the opening of the pylorus into the bulb is more frequently seen with duodenal ulcer than with pyloric.

Carman[25] classified bulbar deformities as: (1) General; (2) basal border; (3) crater; (4) incisural; (5) contracted; (6) accessory pocket; and (7) pseudodiverticula. These correspond to the grouping of Åkerlund,[4] who notes six main types: (1) Niche; (2) transverse encroachments; (3) longitudinal restrictions (such as shortening of the bulb); (4) pouch formation (due to sacculation); (5) pseudodiverticula; and (6) relief deformities (such as due to spasm and hypertrophy of the muscularis mucosa).

In the presence of an ulcer the duodenal bulb is very apt to be irritable and hyperactive, emptying rapidly and often rendering visualization difficult. Reflexly gastric peristalsis, tone and motility are all apt to be increased (Carman,[25] Golden,[63] Holmes and Ruggles,[71] Köhler[87]). In consequence, gastric emptying may be very rapid. However, if the ulcer crater is near the pylorus a marked pylorospasm may result, and delay in emptying be noted (Golden[63]). Such spasm and obstruction due to actual scarring in the bulb account for the retention noted in the stomachs of duodenal ulcer patients. As discussed under gastric ulcers, this is usually less marked than in lesions of the stomach, less than 50 per cent of cases showing a six hour retention (Golden[63]).

Duodenal stasis has been noted frequently in association with duodenal ulcer and Friedenwald and Feldman[58] urged the exclusion of such a lesion in the presence of this finding. Duodenal ulcer accounted for delay in emptying of the second portion in 44 per cent of 80 instances of "puddling."

Tenderness definitely localized to the bulb may or may not be present (Åkerlund,[4] Golden[63]).

Some of the sources of error in the roentgenologic diagnosis of the stomach and duodenum have been suggested in the foregoing paragraphs.

Others include the presence of hypertrophied folds, particularly the prepyloric fold, which has led to a false diagnosis of crater (Kirklin,[85] Golden[63]). Irregularities of the entire upper gastro-intestinal tract may be found in association with a closely approximated loop of colon, with ascites, pregnancy and intra-abdominal tumors (Kirklin[85]). A crater may be plugged by mucus, blood, food, or hypertrophied and edematous folds in the duodenum as well as in the stomach (Åkerlund,[4] Cordiner and Calthrop[34]). Small anterior or posterior wall duodenal ulcers may fail of detection as well as those in the distal bulb (Kirklin[84]). In duodenitis a fleeting glimpse of the bulb may cause an ulcer to be overlooked (Kirklin[84]). In this connection Garland[61] stated that in a roentgenologically negative bulb associated with positive clinical symptoms, if the symptoms are acute an ulcer may have been missed; and if they are chronic, they probably did not have an ulcer as their cause. When one of two ulcers or lesions is seen, an incomplete diagnosis may result. Pyloric block, so complete that the pylorus or the bulb cannot be properly visualized, may allow no more than the impression of "pyloric obstruction."

The stomach is readily responsive to irregularities of the nervous system and the endocrines, as well as toxic states and infections in remote organs. As a result, it must be with caution that physiologic changes are interpreted as evidence by themselves, of intrinsic disease (Eusterman[51]). Boyd[21] describes the stomach as weeping for its neighbors, and often so overdoing it as to obscure the basic source of trouble.

Despite these potential sources of error the accuracy with which the trained roentgenologist detects duodenal and gastric lesions is very great. Carman[26] reports accurate diagnoses in 96 per cent of peptic ulcers. Emery and Monroe[48] in 93 per cent, and Blackford and Dwyer[17] in 83 per cent. Smithies'[138] accuracy was 85 per cent in gastric cases, while that of Miller, Pendergrass and Andrews[104] was 94 per cent in duodenal and 88 per cent in gastric.

Table VII records the roentgenologic findings in this series. These cases were examined by workers of greatly varying experience in roentgenologic methods. There did not appear to be any association between the position of the stomach and the presence of ulcer. Irregular, sluggish and absent peristalsis was more frequently recorded in association with gastric than with duodenal ulcers, but this disturbance in physiology was not consistent enough.

In accord with the findings of others, retention was more marked in patients with gastric ulcers than in the duodenal or combined ulcer groups. This was also true of delay in emptying as seen fluoroscopically. When retention was found, the portion of the motor meal, which was present after six hours, was greatest in the combined ulcer cases, being almost twice the amount estimated in the other groups. In this connection it is of interest that pylorospasm was noted less frequently in the gastric group, indicating that such an obstruction probably does not account for retention as often as disturbed motility and peristalsis.

A lesion was seen in 320 (98 per cent) of the 326 cases examined roent-

TABLE VII

RÉSUMÉ AND ANALYSIS OF ROENTGENOLOGIC FINDINGS

	Medical Pathology			Surgical Pathology			Celiotomy			Totals		
	G.U.	D.U.	Comb.	G.U.	D.U.	Comb.	G.U.	D.U.	Comb.	G.U.	D.U.	Comb.
Roentgenologic Findings												
Number of Cases	23 (30%)*	27 (34%)*	10 (52%)*	35 (80%)*	41 (68%)*	8 (73%)*	17 (45%)*	154 (85%)*	11 (85%)*	75 (47%)*	222 (70%)*	29 (68%)*
Position of Stomach												
High	3 (13%)†	5 (18%)†	3 (30%)†	2 (6%)†	6 (15%)†	2 (25%)†	3 (18%)†	12 (8%)†	3 (27%)†	8 (11%)†	23 (15%)†	8 (27%)†
Low	4 (17%)	8 (30%)	1 (10%)	12 (32%)	12 (29%)	2 (25%)	2 (11%)	38 (25%)	5 (45%)	18 (24%)	58 (27%)	8 (27%)
Medium	6 (26%)	14 (52%)	6 (60%)	21 (60%)	24 (56%)	4 (50%)	12 (71%)	104 (68 %)	3 (27%)	39 (52%)	141 (64%)	13 (45%)
Peristalsis												
Normal	9 (39%)†	12 (45%)†	4 (40%)†	11 (31%)†	15 (37%)†	2 (25%)†	4 (24%)†	70 (46%)†	4 (36%)†	24 (33%)†	97 (44%)†	10 (33%)†
Delayed	4 (17%)	4 (15%)	0 (0%)	2 (6%)	5 (12%)	1 (13%)	3 (18%)	10 (7%)	2 (18%)	9 (2%)	19 (9%)	3 (10%)
Irregular	6 (2%)	5 (18%)	4 (40%)	13 (44%)	17 (41%)	5 (63%)	7 (41%)	38 (25%)	2 (18%)	26 (36%)	60 (27%)	11 (38%)
Sluggish	5 (2%)	3 (11%)	3 (30%)	12 (32%)	8 (20%)	4 (50%)	5 (29%)	24 (16%)	4 (36%)	22 (31%)	35 (16%)	11 (38%)
Hyperactive	4 (17%)	4 (15%)	4 (40%)	2 (6%)	7 (17%)	0 (0%)	2 (12%)	16 (10%)	2 (18%)	8 (11%)	27 (12%)	6 (21%)
Constricting	0 (0%)	1 (4%)	0 (0%)	1 (3%)	3 (7%)	0 (0%)	0 (0%)	5 (3%)	0 (0%)	1 (1%)	9 (4%)	0 (0%)
Ineffective	1 (4%)	3 (11%)	0 (0%)	0 (0%)	1 (2%)	0 (0%)	0 (0%)	6 (4%)	3 (27%)	1 (1%)	10 (5%)	3 (10%)
Reversed	0 (0%)	1 (4%)	0 (0%)	4 (12%)	3 (7%)	2 (25%)	0 (0%)	8 (5%)	1 (9%)	4 (5%)	12 (5%)	3 (10%)
Absent	1 (4%)	1 (4%)	1 (10%)	2 (6%)	0 (0%)	0 (0%)	2 (12%)	5 (3%)	0 (0%)	5 (7%)	6 (3%)	1 (3%)
Gigantic	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	3 (2%)	0 (0%)	0 (0%)	3 (1%)	0 (0%)
Emptying												
Normal	7 (30%)	16 (59%)	3 (30%)	14 (40%)	25 (61%)	3 (38%)	4 (24%)	76 (49%)	3 (27%)	25 (33%)	117 (53%)	9 (31%)
Delayed	5 (22%)	6 (22%)	3 (30%)	17 (49%)	12 (29%)	3 (38%)	12 (71%)	66 (43%)	7 (64%)	34 (45%)	84 (38%)	13 (45%)
Slow	0 (0%)	1 (4%)	0 (0%)	0 (0%)	1 (2%)	0 (0%)	0 (0%)	4 (3%)	0 (0%)	0 (0%)	6 (3%)	0 (0%)
Rapid	3 (13%)	0 (0%)	0 (0%)	3 (9%)	1 (2%)	0 (0%)	0 (0%)	2 (1%)	0 (0%)	6 (8%)	3 (1%)	0 (0%)
Little	0 (0%)	1 (4%)	0 (0%)	1 (3%)	2 (5%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	1 (1%)	3 (1%)	0 (0%)
None seen	0 (0%)	1 (4%)	2 (20%)	1 (3%)	0 (0%)	1 (13%)	4 (24%)	2 (1%)	1 (9%)	5 (7%)	3 (1%)	4 (14%)
Retention												
Cases showing	11 (48%)	9 (33%)	4 (40%)	19 (54%)	16 (39%)	3 (38%)	16 (94%)	69 (45%)	6 (55%)	46 (61%)	94 (42%)	13 (45%)
Average percentage of 6 hr. meal retention	41%	67%	83%	35%	48%	24%	38%	36%	75%	36%	41%	66%
Local Findings												
Stomach												
Pylorospasm	7 (30%)†	10 (37%)†	5 (50%)†	7 (20%)†	8 (20%)†	2 (25%)†	2 (12%)†	32 (21%)†	2 (18%)†	16 (21%)†	50 (21%)†	9 (31%)†
Exaggerated mucosal folds	2 (9%)	4 (15%)	2 (20%)	6 (17%)	3 (7%)	1 (13%)	3 (18%)	8 (5%)	2 (18%)	11 (14%)	15 (7%)	5 (17%)
Crater	14 (60%)	1 (4%)	1 (10%)	27 (78%)	2 (5%)	2 (25%)	12 (71%)	1 (1%)	5 (45%)	53 (71%)	4 (2%)	8 (28%)
Incisura	6 (26%)	0 (0%)	2 (20%)	8 (23%)	3 (7%)	0 (0%)	1 (6%)	0 (0%)	0 (0%)	15 (20%)	3 (1%)	2 (7%)
Irregularity	1 (4%)	0 (0%)	2 (20%)	0 (0%)	2 (5%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	1 (1%)	2 (1%)	2 (7%)
Filling defect	4 (17%)	0 (0%)	0 (0%)	9 (26%)	1 (2%)	1 (13%)	5 (29%)	0 (0%)	0 (0%)	18 (24%)	1 (0.5%)	1 (3%)

Bulb												
Spasm	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	1 (6%)	0 (0%)	0 (0%)	1 (1%)	0 (0%)	0 (0%)
Deformity	5 (22%)	20 (74%)	5 (50%)	1 (3%)	23 (56%)	3 (38%)	2 (12%)	9 (6%)	8 (73%)	8 (11%)	52 (23%)	16 (55%)
Irregularity	4 (17%)	4 (15%)	2 (20%)	3 (9%)	15 (37%)	2 (25%)	1 (6%)	42 (27%)	0 (0%)	8 (11%)	61 (27%)	4 (14%)
Irritability	1 (4%)	0 (0%)	1 (10%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	9 (6%)	0 (0%)	1 (1%)	9 (4%)	1 (3%)
Not seen	2 (9%)	1 (4%)	2 (20%)	2 (6%)	2 (5%)	0 (0%)	1 (6%)	7 (5%)	1 (9%)	5 (7%)	10 (5%)	3 (10%)
Crater	0 (0%)	6 (22%)	4 (40%)	3 (9%)	8 (20%)	1 (13%)	0 (0%)	40 (26%)	0 (0%)	3 (4%)	54 (24%)	5 (17%)
Duodenum												
Dilated 2nd portion	0 (0%)	0 (0%)	0 (0%)	5 (14%)	3 (7%)	0 (0%)	0 (0%)	8 (5%)	1 (9%)	5 (7%)	11 (5%)	1 (3%)
Duodenitis	0 (0%)	2 (7%)	0 (0%)	1 (3%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	1 (1%)	2 (1%)	0 (0%)
Location of Lesion												
Ant. wall	0 (0%)	1 (4%)	0 (0%)	2 (6%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	2 (2%)	1 (0.5%)	0 (0%)
Post. wall	4 (17%)	0 (0%)	0 (0%)	5 (14%)	0 (0%)	0 (0%)	2 (12%)	0 (0%)	0 (0%)	12 (16%)	0 (0%)	0 (0%)
Lesser curv.	10 (43%)	0 (0%)	2 (20%)	20 (57%)	2 (5%)	1 (13%)	10 (59%)	1 (6%)	3 (27%)	40 (54%)	3 (1%)	6 (21%)
Greater curv.	5 (22%)	0 (0%)	0 (0%)	3 (9%)	0 (0%)	1 (13%)	0 (0%)	0 (0%)	1 (9%)	8 (11%)	0 (0%)	2 (7%)
Perforation Diagnosed	0 (0%)‡	1 (5%)‡	1 –	1 (20%)‡	1 (10%)‡	0 (0%)‡	5 (23%)‡	2 (5%)‡	1 (9%)‡	6 (16%)‡	4 (6%)‡	2 –
SUMMARY												
Ulcers with correct diagnosis	18 (78%)§	22 (81%)§	5 (20%)§	26 (69%)§	41 (100%)§	5 (63%)§	16 (94%)§	149 (97%)§	6 (46%)§	60 (80%)§	212 (95%)§	16 (55%)§
Ulcers with incorrect diagnosis	5 (22%)	5 (19%)	5 (50%)	9 (31%)	0 (0%)	3 (37%)	1 (6%)	5 (3%)	5 (54%)	15 (20%)	10 (5%)	13 (45%)
					Combined Accuracy 88%							
Type of Error												
Not seen or incorrectly interpreted	2 (40%)||	3 (60%)||	0 (0%)||	3 (33%)||	0 (0%)||	0 (0%)||	0 (0%)||	5 (100%)||	0 (0%)||	5 (33%)||	8 (80%)||	0 (0%)||
Duod. and gast. only 1 seen	0 (0%)	0 (0%)	5 (100%)	0 (0%)	0 (0%)	2 (67%)	0 (0%)	0 (0%)	5 (100%)	0 (0%)	0 (0%)	12 (92%)
Duod. called gastric	0 (0%)	1 (20%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	1 (10%)	0 (0%)
Gastric called duodenal	1 (20%)	0 (0%)	0 (0%)	2 (22%)	0 (0%)	0 (0%)	1 (100%)	0 (0%)	0 (0%)	4 (27%)	0 (0%)	0 (0%)
Carc. diag., ulcer found	1 (20%)	0 (0%)	0 (0%)	4 (44%)	0 (0%)	1 (33%)	0 (0%)	0 (0%)	0 (0%)	5 (33%)	0 (0%)	1 (8%)
Ulcer diag., carc. found	1 (20%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	1 (6%)	0 (0%)	0 (0%)
Gastric and duod. diag., one found	0 (0%)	1 (20%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	1 (10%)	0 (0%)
Multiple ulcers diag. and one found	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)

* Percentage of cases with ulcer.

† Percentage of cases examined roentgenologically.

‡ The percentage represents the per cent of the cases which perforated.

§ The percentage represents the degree of accuracy.

|| The percentage represents the per cent of those cases in which the diagnosis was in error.

genologically. The finding of a crater led to the diagnosis of gastric ulcer in 53 of the 60 cases correctly interpreted. Of the remaining seven instances, three were prepyloric ulcers in which antral spasm led to the diagnosis although no crater was seen. Two were "hour-glass" stomachs in which the crater could not be demonstrated. One was a healed ulcer which had caused a defect or stiffness in the gastric contour; and the last was an ulcer in the pylorus, causing complete obstruction and retention. Of the duodenal ulcers, a crater was seen in 24 per cent of instances. This is the usual figure where no special technics are used. In all other patients with duodenal ulcer the roentgenologic diagnosis was made when bulbar deformity was seen.

Two hundred eighty-eight of the 326 cases were accurately diagnosed (88 per cent). Ninety-five per cent of the duodenal cases were correct, 80 per cent of the gastric, and 55 per cent of the combined. About one-third of all the errors fell in this last group, 13 cases in all. In 12 of these the error was in failure to see both lesions. Ten of the 12 "missed" ulcers were gastric, and craters could be seen on reviewing the roentgenograms in nine instances. The other two "missed" ulcers were duodenal, one of which had caused bulbar deformity which was seen on reviewing the films. The other error in the combined group was in a case in which the ulcer involved the pylorus, an impression that the lesion was malignant having been given.

Three gastric and three duodenal ulcers were not seen. One gastric diverticulum, and the markedly hypertrophied folds associated with ulcer, were considered as ulcer and polypi respectively. In the bulb one ulcer was called a diverticulum and another was attributed to periduodenal adhesions. A third case had a deformed bulb due to an ulcer which was thought to be due to external pressure. The remaining errors shown in Table VII are self-explanatory.

SUMMARY.—An analysis of 520 ulcer patients has been presented. Insofar as possible comparable findings from similar material examined by others has been recorded, and the opinions of the writers noted. Comment has been made, where possible, on the association of the clinical, pathologic and roentgenologic findings.

It is recognized that this group of cases is selective and cannot, therefore, be considered as giving "typical" statistics. This shortcoming, however, is somewhat overbalanced in that the cases are all "proven." In order that the source of each finding may be judged, the tables include the results of the analyses of autopsy and operated cases.

CONCLUSIONS

It would seem, from this study, that:

(1) Statistics relative to peptic ulcer vary considerably, due to the source of the material and the interpretation of the author.

(2) Ulcers arise in an area of alimentary tract mucosa where the resistance has been lowered and where gastric juice may exert an injurious effect.

(3) Peptic ulcers occur in from 1 to 2 per cent of the population as a

whole, and in about the same frequency in general hospital admissions. They are encountered about three times as often as this in postmortem studies.

(4) Both duodenal and gastric ulcers are more frequent in men than in women.

(5) There is no racial selectivity.

(6) Patients with gastric ulcer averaged older than those with duodenal ulcer, and the peak incidence was between 40 and 50 years of age.

(7) Duodenal ulcers are more frequent than gastric. Acute ulcers are more frequent in the stomach than in the duodenum, which may account for the discrepancy between clinical and pathologic statistics.

(8) Ulcers of both the stomach and the duodenum are present in about 10 per cent of ulcer patients.

(9) Pathologically, gastric and duodenal ulcers are essentially similar.

(10) Benign gastric ulcers are more frequent on the lesser curvature, within 5 cm. of the pylorus.

(11) Duodenal ulcers are more frequent within 1 cm. of the pylorus, and are practically limited to the bulb.

(12) Gastric ulcer craters are usually less than 3 cm. in diameter and those of duodenal ulcers are usually under 1 cm.

(13) Perforation occurs in about 5 to 10 per cent of hospitalized peptic ulcer cases.

(14) Hemorrhage of some degree is present in about 20 per cent of hospitalized ulcer patients and in 10 per cent of all ulcer cases.

(15) Carcinoma arises in gastric ulcer in 5 to 10 per cent of cases.

(16) The mortality due directly to peptic ulcers is about 2 to 5 per cent.

(17) No direct association between disease of the gallbladder or appendix and peptic ulcer could be demonstrated.

(18) The symptomatology of peptic ulcer is marked by remissions and exacerbations, and is subject to seasonal variations.

(19) Pain, the most important symptom of peptic ulcer, bears a definite relationship to eating. It occurs earlier in gastric than in duodenal lesions.

(20) Local tenderness is the most constant physical finding in peptic ulcer patients.

(21) The diagnosis of duodenal ulcer roentgenologically depends upon the demonstration of a definite crater or a deformed bulb. The diagnosis of gastric ulcer depends upon the demonstration of a crater and of abnormal physiologic behavior or deformed anatomic structure.

(22) Retention after six hours is more frequent in the presence of gastric than of duodenal ulcers, although the amount of retention is slightly greater in the latter. Gastric ulcers which cause retention may be remote from the pyloric ring.

(23) Gastric peristalsis is apt to be hyperactive in the presence of a duodenal ulcer, and gastric tone and motility may be increased.

(24) Duodenal ulcer causes spasm and scarring in the bulb, and the deformity so produced is almost always due to such a lesion.

BIBLIOGRAPHY

[1] Aaron, C. D.: Chronic Appendicitis, Pylorospasm and Duodenal Ulcer. J.A.M.A., **64**, 1845, 1915.

[2] Åkerlund, Å.: Later Experiences Concerning the Niche Diagnosis in Cases of Duodenal Ulcer. Acta Radiol., **8**, 538, 1927.

[3] Åkerlund, Å.: The Roentgen Diagnosis of Ulcus Duodeni with Respect to the Local "Direct" Roentgen Symptoms. Acta Radiol., **2**, 14, 1923.

[4] Åkerlund, Å.: The Present Day Criteria of X-ray Diagnosis of Duodenal Ulcer. Am. Jour. Surg., **11**, 233 and 504, 1931.

[5] Albrecht, H. U.: Das ulcus problem im lichte moderner röntgenforschung. Leipsic, Georg Thieme, 1930.

[6] Alvarez, W. C., and MacCarty, W. C.: Sizes of Resected Gastric Ulcers and Gastric Carcinomas. J.A.M.A., **91**, 226, 1928.

[7] Alvarez, W. C.: What Is the Risk of Insuring Applicants with Peptic Ulcer? Am. Jour. Med. Sci., **178**, 177, 1929.

[8] Alvarez, W. C., Horowitz, A., and Ascanio, H.: The Normal Thickness of the Pyloric Muscle and the Influence on It of Ulcer, Gastro-Enterostomy and Carcinoma. ANNALS OF SURGERY, **89**, 521, 1929.

[9] Balfour, D. C.: Proc. Staff Meet., Mayo Clin., **6**, 55, 1931.

[10] Balfour, D. C.: Results of Gastro-Enterostomy for Ulcer of the Duodenum and Stomach. ANNALS OF SURGERY, **92**, 558, 1930.

[11] Balfour, D. C.: Surgical Treatment of Hemorrhagic Duodenal Ulcer. ANNALS OF SURGERY, **96**, 581, 1932.

[12] Barclay, A. E.: The Digestive Tract. Cambridge University Press, 1933.

[13] Beaver, M. D., and Mann, F. C.: The Production of Peptic Ulcer after Section of the Gastric Nerve. ANNALS OF SURGERY, **94**, 1116, 1931.

[14] Berg, H. H.: The Direct Signs of Duodenal Ulcer. Brit. Jour. Radiol., **30**, 372, 1925.

[15] Berkman, D. M.: Gastric Retention: Its Treatment and the Surgical Mortality. Coll. Papers Mayo Clin., **15**, 92, 1932.

[16] Bevan, A. D.: The Pathologic Anatomy and Physiology of Peptic Ulcer. Surg. Clin. N. Amer., **14**, 979, 1934.

[17] Blackford, J. M., and Dwyer, M. F.: A Clinical Review of 250 Case Histories of Peptic Ulcer with Particular Reference to Age, Duration of Symptoms and Complications. Tr. Am. Gastro-enterol. Assn., **29**, 112, 1927.

[18] Blahd, E. M.: Peptic Ulcer, A Statistical Study. Ohio State Med. Jour., **30**, 156, 1934.

[19] Bloomfield, A. L.: The Diagnosis of Early Cancerous Changes in Peptic Ulcer. J.A.M.A., **104**, 1197, 1935.

[20] Bolton, C.: Ulcer of the Stomach. London, Edward Arnold and Company, 1913.

[21] Boyd, W.: Surgical Pathology. Philadelphia, W. B. Saunders Company, 1933 (3rd Edition).

[22] Braithwaite, L. R.: The Flow of the Lymph from the Ileocecal Angle and Its Possible Bearing on the Cause of Duodenal and Gastric Ulcer. Brit. Jour. Surg., **11**, 7, 1923.

[23] Cabot, H., and Adie, G. C.: The Etiology of Cancer of the Stomach. A Review of One Hundred Sixteen Consecutive Cases of Cancer of the Stomach with Particular Relation to Etiology. ANNALS OF SURGERY, **82**, 86, 1925.

[24] Carman, R. D.: The Duodenal Niche. Am. Jour. Roentgenol., **16**, 101, 1926.

[25] Carman, R. D.: Roentgen Diagnosis of Disease of the Alimentary Tract. Philadelphia, W. B. Saunders Company, 1917.

[26] Carman, R. D.: The Roentgen Diagnosis and Localization of Peptic Ulcer. California State Jour. Med., **18**, 377, 1920.

[27] Cecil, R. L.: Text book of Medicine. Philadelphia, W. B. Saunders Company, 1929 (1st Edition).

[28] Chace, A. F.: The Medical Aspects of Peptic Ulcer with Special Reference to Diagnosis and Treatment. Am. Jour. Digest. Dis. and Nutrit., **1**, 866, 1934.

[29] Chang, H. C.: The Incidence of Malignancy in Gastric Ulcer. Am. Jour. Digest. Dis. and Nutrit., **3**, 10, 1936.

[30] Clairmont, P.: Die pathologisch-anatomischen veränderungen des duodenums bie ulkus und deren darstellungen im röntgenbilde. Wurzb. Abhandl. a. d. Gesamtgeb. d. Med., **1**, 1923.

[31] Clark, D. M., and Geyman, M. J.: The Roentgen Evidence of Healing in Duodenal Ulcer. J.A.M.A., **102**, 107, 1934.

[32] Cleland, J. B.: Carcinoma of the Stomach, Gastric Ulcers and Duodenal Ulcers in One Thousand Consecutive Autopsies at the Adelaide Hospital. Med. Jour. Australia, **14**, 740, 1927.

[33] Cole, L. G.: The Diagnosis of Post-pyloric (Duodenal) Ulcer by Means of Serial Radiography. Lancet, **1**, 1239, 1914.

[34] Cordiner, G. R., and Calthrop, G. T.: The Radiography of the Duodenal Cap. Brit. Jour. Radiol., **23**, 700, 1936.

[35] Corria, C. M. R.: Hyperplasia of Pyloric Glands (Gastric Brunnerian Formations); A Case in Relation to Gastric Ulcer. Rev. Soc. argent. de biol., **2**, 56, 1926; (Abs. J.A.M.A., **87**, 886, 1926).

[36] Crohn, B. B.: Affections of the Stomach. Philadelphia, W. B. Saunders Company, 1927.

[37] Curling, T. B.: On Acute Ulceration of the Duodenum in Cases of Burns. Tr. Med.-Chir. Soc. London, **25**, 260, 1842.

[38] Cushing, H.: Peptic Ulcers and the Interbrain. Surg., Gynec., and Obst., **55**, 1, 1932.

[39] Deaver, J. B.: Chronic Duodenal Ulcer. Surg., Gynec., and Obst., **50**, 745, 1930.

[40] Deaver, J. B., and Burden, V. G.: The Pyloric Sphincter and Duodenal Ulcer. ANNALS OF SURGERY, **94**, 818, 1931.

[41] DeLario, A. J.: A Roentgenologic Follow-Up of 125 Cases of Peptic Ulcer with Clinical and Laboratory Findings. Am. Jour. Roentgenol., **34**, 190, 1935.

[42] de la Viesca, P.: Die grosse magenblutung. Arch. f. Verdauungskr., **58**, 22, 1935.

[43] Diamond, J. S.: The Niche as a Roentgen Sign of Duodenal Ulcer with a Report of 42 Cases. Radiology, **4**, 93, 1925.

[44] Dible, J. H.: Gastric Ulcer and Gastric Carcinoma: An Inquiry into Their Relationship. Brit. Jour. Surg., **12**, 666, 1925.

[45] Dragstedt, L. R.: Contributions to the Physiology of the Stomach: Gastric Juice in Duodenal and Gastric Ulcers. J.A.M.A., **68**, 330, 1917.

[46] Eggers, H.: Gastritis und konstitutionelle innervations und zirkulations-storungen. Deutsche Med. Wchnschr., **55**, 957, 1929.

[47] Eggleston, E. L.: A Critical Review of Five Hundred Cases of Gastric and Duodenal Ulcer. J.A.M.A., **75**, 1542, 1920.

[48] Emery, E. S., Jr., and Monroe, R. T.: Peptic Ulcer, A Study of 556 Cases. Arch. Int. Med., **43**, 846, 1929.

[49] Ettinger, A., and Davis, W. E.: The X-ray Diagnosis of the Activity and the Cure of Duodenal Ulcer. Am. Jour. Digest. Dis. and Nutrit., **1**, 579, 1934.

[50] Eusterman, G. B.: The Incidence and Diagnosis of Complicating Factors in Gastric and Duodenal Lesions. Results in 1,800 Cases Operated Upon. Am. Jour. Gastro-enterol., **3**, 111, 1913.

[51] Eusterman, G. B.: The Diagnostic Aspects of Roentgenologically Negative Gastric Disorders. J.A.M.A., **107**, 1432, 1936.

[52] Eusterman, G. B., and Balfour, D. C.: The Stomach and Duodenum. Philadelphia, W. B. Saunders Company. 1935.

[53] Ewing, J.: The Relation of Gastric Ulcer to Gastric Carcinoma. ANNALS OF SURGERY, **67**, 715, 1918.

[54] Forssell, G.: Studies of the Mechanism of Movement of the Mucous Membrane of the Digestive Tract. Am. Jour. Roentgenol., **10,** 87, 1923.
[55] Fraenkel, A.: Certain Dynamic Radiological Signs Found in Association with Gastric Ulcer. Brit. Jour. Radiol., **32,** 45, 1927.
[56] Fraenkel, A.: The Cross-Bar Symptom. Brit. Jour. Radiol., **31,** 349, 1926.
[57] Friedenwald, J., and Love, W. S.: Raynaud's Disease Complicated with Gastric Ulcer—Case Report. J.A.M.A., **85,** 83, 1925.
[58] Friedenwald, J., and Feldman, M.: The Incidence of Peptic Ulcer Associated with Duodenal Stasis. Radiology, **26,** 595, 1936.
[59] Gaither, E.: Peptic Ulcer. New York. Thomas Nelson & Sons, Loose Leaf Medicine, Vol. 5.
[60] Gallagher, W. J.: Acute Traumatic Ulcers of the Small Intestine. Arch. Surg., **15,** 689, 1927.
[61] Garland, L. H.: The Roentgen Diagnosis of Duodenal Ulcer. Radiology, **14,** 482, 1930.
[62] George, A. W., and Gerber, I.: The Direct Method of Diagnosis of Duodenal Ulcer by Means of the Roentgen-ray. Am. Jour. Roentgenol., **1,** 287, 1914.
[63] Golden, R.: The Roentgen-ray Examination of the Digestive Tract. New York, Thomas Nelson and Sons. Diagnostic Roentgenology, 1936.
[64] Goldman, L.: Gross Hemorrhage from Peptic Ulcer; Its Morbidity, Mortality and Treatment. J.A.M.A., **107,** 1537, 1936.
[65] Hartman, H. R., and Rivers, A. B.: Benign Gastric and Duodenal Ulcer: Clinical Diagnosis and Conditions found at Operation. Arch. Int. Med., **44,** 314, 1929.
[66] Hartzell, J. B.: The Effect of Section of the Vagus Nerves on Gastric Acidity. Am. Jour. Physiol., **91,** 161, 1929.
[67] Haudek, M.: Der radiologische nachweis des ulcus duodeni. Med. Klin., **8,** 181, 1912.
[68] Hauser, G., Henke, F., and Lubarsch, O.: System of Pathological Anatomy and Histology.
[69] Hinton, J. W., and Trabek, M.: The Transformation of Gastric Ulcer into Gastric Carcinoma. Surg., Gynec., and Obst., **64,** 16, 1937.
[70] Hinton, J. W.: A Review of 746 Gastric and Duodenal Ulcers. Am. Jour. Digest. Dis. and Nutrit., **3,** 59, 1936.
[71] Holmes, G. W., and Ruggles, H. E.: Roentgen Interpretation. Philadelphia, Lea and Febiger, 1936.
[72] Holmes, G. W., and Hampton, A. O.: The Importance of Location in the Differential Diagnosis of Benign and Malignant Gastric Ulcerations. New England Jour. Med., **208,** 971, 1933.
[73] Holmes, G. W., and Schatzki, R.: The Examination of the Mucosal Relief as a Diagnostic Aid in Diseases of the Gastro-intestinal Tract. Am. Jour. Roentgenol., **34,** 145, 1935.
[74] Hunt, T.: Chronic Peptic Ulcer. Practitioner, **138,** 139, 1937.
[75] Hurst, A. F., and Ryle, J. A.: The Incidence, Mortality and Treatment of Hemorrhage in Gastric and Duodenal Ulcer. Lancet, **231,** 1, 1937.
[76] Hurst, A. F., and Stewart, M. J.: Gastric and Duodenal Ulcer. London, Oxford University Press. 1929.
[77] Hurst, A. F.: The Sensibility of the Alimentary Canal. Oxford Medical Publications. 1911.
[78] Jefferson, G.: Carcinoma of the Suprapapillary Duodenum Causally Associated with Pre-existing Simple Ulcer. Brit. Jour. Surg., **4,** 209, 1916.
[79] Jordan, S.: A Review of the Gastric Ulcer Problem. J.A.M.A., **107,** 1451, 1936.
[80] Judin, S. S.: Partial Gastrectomy in Acute Perforated Peptic Ulcer. Surg., Gynec., and Obst., **64,** 63, 1937.
[81] Kaiser, F. J.: Mechanical Factors in the Origin of Gastric Ulcer. Arch. f. klin. Chir., **134,** 535, 1925; (Abst. J.A.M.A., **84,** 1310, 1925).

[82] Karsner, H. T.: Human Pathology. Philadelphia, J. B. Lippincott Company, 3rd Edit., 1931.

[83] Kerley, P.: Recent Advances in Radiology. Philadelphia, P. Blakiston's Son and Company. 2nd Edit., 1936.

[84] Kirklin, B. R.: Duodenal Ulcers that May Escape Roentgenologic Diagnosis. Med. Clin. N. Amer., **15,** 177, 1931.

[85] Kirklin, B. R.: Factors of Error in the Roentgenologic Distinction between the Normal and the Diseased Stomach and Duodenum. Am. Jour. Digest. Dis. and Nutrit., **1,** 260, 1934.

[86] Kirklin, B. R., and Burch, H. A.: The Incidence and Significance of the Niche in Duodenal Ulcer. Ann. Int. Med., **9,** 436, 1935.

[87] Köhler, A.: Roentgenology. New York, William Wood and Company, 1935.

[88] Konjetzny, G. E.: Die chronische gastritis des ulcus magens, Zentralbl. f. Chir., **50,** 1026, 1923.

[89] Laird, E. G.: The Co-incidence of Cholecystitis and Peptic Ulcer. New England Jour. Med., **213,** 764, 1935.

[90] Landon, L. H.: The Gastric-Ulcer-Cancer Problem. ANNALS OF SURGERY, **101,** 536, 1935.

[91] Larimore, J. W.: Duodenal and Gastric Ulcer, Cholecystitis and Appendicitis: A Consideration of Their Pathological Relations. Surg., Gynec., and Obst., **50,** 59, 1930.

[92] Lehmann, H.: Frequency and Localization of Peptic Ulcer in the Stomach and the Duodenum. Mitt. a. d. Grenzgeb. d. Med. u. Chir., **35,** 185, 1926; (Abst. J.A.M.A., **87,** 888, 1926).

[93] Lindau, A., and Wulff, H.: The Peptic Genesis of Gastric and Duodenal Ulcers; Especially in the Light of Ulcers in Meckel's Diverticulum and the Postoperative Ulcers in the Jejunum. Surg., Gynec., and Obst., **53,** 621, 1931.

[94] Lynch, R.: An Analysis of Ulcer of the Stomach and Duodenum Based on a Study of 944 Case Reports. Canad. Med. Assn. Jour., **17,** 677, 1927.

[95] MacGuire, D. P.: Bleeding Gastric and Duodenal Ulcers. Am. Jour. Digest. Dis. and Nutrit., **2,** 430, 1935.

[96] Maingot, G.: Tableau synoptique des rétentions gastriques. Rev. de Med., Paris, **45,** 645, 1928.

[97] Mann, F. C.: The Chemical and Mechanical Factors in Experimentally Produced Peptic Ulcer. Surg. Clin. N. Amer., **5,** 753, 1925.

[98] Martin, E., and Burdin, V. G.: Pyloric Achalasia and Peptic Ulcer. ANNALS OF SURGERY, **88,** 565, 1928; (Also Tr. Am. Surg. Assn., **46,** 249, 1928).

[99] Matthews, W. B.: The Production of Intestinal Ulcers by Active Gastric Juice. Proc. Soc. Exper. Biol. and Med., **28,** 960, 1931.

[100] Matthews, W. B., and Dragstedt, L. R.: The Etiology of Gastric and Duodenal Ulcers. Surg., Gynec., and Obst., **55,** 265, 1932.

[101] Mayo, W. J.: Pathologic Data Obtained from Ulcers Excised from the Anterior Wall of the Duodenum. ANNALS OF SURGERY, **57,** 691, 1913.

[102] Meisser, J. G.: Further Studies on the Elective Localization of Bacteria from Infected Teeth. Jour. Am. Dent. Assn., **12,** 554, 1925.

[103] Miller, R. H.: A Present Day Review of Gastric and Duodenal Ulcer. New England Jour. Med., **206,** 925, 1932.

[104] Miller, T. G., Pendergrass, E. P., and Andrews, K. S.: A Statistical Study of Clinical and Laboratory Findings in Gastric and Duodenal Ulcer with Special Reference to Roentgenologic Data. (Based on records of 279 operatively demonstrated cases.) Am. Jour. Med. Sci., **177,** 15, 1933.

[105] Morley, J.: The Relation of Gastric Ulcer to Carcinoma. Lancet, **2,** 823, 1923.

[106] Morton, C. B.: Observations on Peptic Ulcer. (1) A Method of Producing Chronic Gastric Ulcer: A Consideration of Etiology. ANNALS OF SURGERY, **85,** 207, 1927.

[107] Moszkowicz, L.: Ueber die entstehung der magengeschwurkrankeit. (Gastropathie.) Klin. Wchnschr., **9**, 385, 1930.

[108] Moynihan, Lord B.: Two Lectures on Gastric and Duodenal Ulcer, A Record of Ten Years' Experience. Bristol (Eng.), John Wright and Sons, Ltd., 1923.

[109] Moynihan, Lord B.: Peptic Ulcer, A Review of the Collective Inquiry. Brit. Med. Jour., **1**, 458, 1930.

[110] Orator, V., and Metzler, F.: Klinische und experimentelle beitrage zur ulcus frage. (3) Zur frage der pathogenese und malignen entartung des magen-duodenalgeschwurs. Deutsche Ztschr. f. Chir., **202**, 167, 1927.

[111] Osler, W. (McCrae, T.): The Principles and Practice of Medicine. New York, D. Appleton & Co., 1932. (12th Edit.)

[112] Palmer, W. L.: The Mechanism of Pain in Gastric and Duodenal Ulcer. Arch. Int. Med., **38**, 694, 1926.

[113] Paterson, H. J.: The Treatment of Severe Gastric and Duodenal Hemorrhage. Proc. Roy. Soc. Med., **17**, 1, 1924.

[114] Payr, E.: Experimente über magenveranderungen als folge von thrombose und embolie im pfortadergebeite. Arch. f. klin. Chir., **84**, 799, 1907.

[115] Percy, N. M., and Beilin, D. S.: An Analysis of 1,000 Consecutive Examinations of the Stomach and Duodenum from the Clinical, Roentgenologic and Surgical Viewpoints. Am. Jour. Roentgenol., **32**, 179, 1934.

[116] Peter, G.: The Diagnosis of Duodenal Ulcer. Radiology, **19**, 360, 1932.

[117] Puhl, E.: Ueber die etstchung und entwicklung des magen-duodenalgeschwurs. Arch. f. klin. Chir., **158**, 1, 1930.

[118] Reeves, T. B.: A Study of the Arteries Supplying the Stomach and Duodenum and Their Relation to Ulcer. Surg., Gynec., and Obst., **30**, 374, 1920.

[119] Reiche, F.: Zür diagnose des ulcus ventriculi im röntgenbild. Fortschr. a. d. Geb. d. Röntgenstrahlen, **14**, 171, 1909.

[120] Rivers, A. B.: Pain in Benign Ulcer of the Esophagus, Stomach and Small Intestine. J.A.M.A., **104**, 169, 1935.

[121] Rivers, A. B.: Clinical Consideration of the Etiology of Peptic Ulcer. Arch. Int. Med., **53**, 97, 1934.

[122] Rivers, A. B., and Dry, T. G.: The Differentiation of Benign and Malignant Gastric Ulcers (The Unreliability of Diagnostic Criteria). Arch. Surg., **30**, 702, 1935.

[123] Rivers, A. B., and Mason, J. B.: The Association of Peptic Ulcer and Cholecystic Disease. Minnesota Med., **14**, 330, 1931.

[124] Robertson, H. E., and Hargis, E. H.: Duodenal Ulcer, An Anatomic Study. Med. Clin. N. Amer., **8**, 1065, 1925.

[125] Robinson, S. C.: A Classification of Gastroduodenal Ulcers on the Basis of Their Etiology. Am. Jour. Digest. Dis. and Nutrit., **3**, 375, 1936.

[126] Robinson, S. C.: On the Etiology of Peptic Ulcer. An Analysis of 70 Ulcer Patients. Am. Jour. Digest. Dis. and Nutrit., **2**, 333, 1935.

[127] Roof, C. S.: The Differential Diagnosis of Gastric Ulcer, Duodenal Ulcer and Carcinoma of the Stomach as Found in a Study of 337 Hospital Cases. Jour. Med., **15**, 499, 1934.

[128] Rosenow, E. C.: Experimental and Clinical Studies on Focal Infection and Elective Localization; Newer Findings and Their Significance. Jour. Am. Dent. Assn., **11**, 963, 1924.

[129] Sallick, M.: Late Results in Acute Perforated Peptic Ulcer Treated by Simple Closure. Annals of Surgery, **104**, 853, 1936.

[130] Sauer, P. K.: Carcinoma Following Gastric and Duodenal Ulcer. Ann. Surg., **102**, 995, 1935.

[131] Schinz, Baensch, and Friedl.: Lehrbuch d. Röntgendiagnostik. Leipsic. Georg Thieme. 1934.

[132] Scott, W. J. M.: The Relationship of Carcinoma and Callous Gastric Ulcer. Surg., Gynec., and Obst., **46**, 199, 1928.

[133] Scott, W. J. M.: The Possibility of Malignancy as It Affects the Treatment of Chronic Gastric Ulcer. ANNALS OF SURGERY, **102**, 586, 1935.

[134] Singer, G.: Peptisches geschwur-lungentuberkulose anatomische vaguserkrankung. Arch. f. Verdauungskr., **43**, 410, 1928.

[135] Singleton, A. C.: Benign Prepyloric Ulcer. Radiology, **26**, 198, 1936.

[136] Sippy, B. W.: Gastric and Duodenal Ulcer. Medical Care by an Efficient Removal of Gastric Juice Corrosion. J.A.M.A., **64**, 1625, 1915.

[137] Smithies, F.: On an Adequate Conception of the Etiology and the Significance of Peptic Ulcer (Gastric and Duodenal). Am. Jour. Digest. Dis. and Nutrit., **2**, 437, 1935.

[138] Smithies, F.: Notes on the Diagnosis and Prognosis in Gastric Ulcer. A Clinical Study of 500 Consecutive, Operatively Demonstrated Cases. Am. Jour. Digest. Dis. and Nutrit., **1**, 697, 1934.

[139] Somervell, T. C., and Orr, I. M.: Some Contributions to the Causation, Pathology and Treatment of Duodenal Ulcer and Its Complications. Brit. Jour. Surg., **24**, 227, 1936.

[140] Spilsbury, B. H.: The Morbid Anatomy and Histology of Gastric Ulcer. Proc. Roy. Soc. Med. (Section Med.), **15**, 25, 1922.

[140a] Sproull, J.: Discussion of the Occurrence of Benign Ulcer on the Greater Curvature. Am. Jour. Roentgenol., **25**, 464, 1931.

[141] Steinberg, M. E.: The Stomach Mucosa in Ulcer and Carcinoma. Histologic Studies. Arch. Surg., **14**, 991, 1927.

[142] Stewart, M. J.: The Pathology of Gastric Ulcer; Pathological Observations on Gastric and Duodenal Ulcer. Brit. Med. Jour., **2**, 1021, 1923.

[143] Stewart, M. J.: The General Relation of Carcinoma to Ulcer. Brit. Med. Jour., **1**, 882, 1925.

[144] Stewart, W. H., and Illick, H. E.: Why Peptic Ulcers are Missed on X-ray Examination. Am. Jour. Digest. Dis. and Nutrit., **1**, 52, 1934.

[145] Stone, R. S., and Ruggles, H. E.: The Diagnostic Value of Prepyloric and Pyloric Findings. Am. Jour. Roentgenol., **27**, 193, 1932.

[146] Sturdevant, M., and Shapiro, L. L.: Gastric and Duodenal Ulcer. Arch. Int. Med., **38**, 41, 1926.

[147] Sutherland, C. G.: Duodenal Ulcer: A Comparison of the Roentgenologic and Histologic Findings. Radiology, **8**, 111, 1927.

[148] Twining, E. W.: The Interpretation of Radiographs of the Alimentary Tract. Brit. Med. Jour., **1**, 704, 1936.

[149] Virchow, R.: Historiches, kritisches und positives zur lehre der unterleibsaffektionen. Virchows Arch. f. path. Anat., **5**, 281, 1853.

[150] Von Bergmann, G.: Ulcus duodeni und vegetatives nervensystem. Berl. klin. Wchnschr., **50**, 2374, 1913.

[151] Walton, A. J.: The Surgical Treatment of Simple Ulcers of the Stomach. Lancet, **1**, 1071, 1931.

[152] Wilkie, D. P. D.: Duodenal Ulcer in the Female. The Cholecystoduodenal Syndrome. Lancet, **2**, 1228, 1927.

[153] Wilkie, D. P. D.: The Blood Supply of the Duodenum with Special Reference to the Supraduodenal Artery. Surg., Gynec., and Obst., **13**, 399, 1911.

CHANGING METHODS IN THE SURGICAL TREATMENT OF PEPTIC ULCER*

A STUDY OF THE CASES OPERATED UPON AT THE ROOSEVELT HOSPITAL, NEW YORK

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NEW YORK

FROM THE SURGICAL SERVICES OF THE ROOSEVELT HOSPITAL, NEW YORK

IN NO field of surgery has there been displayed a greater interest than in that of the treatment of ulcers of the stomach and duodenum, and in none, during recent years particularly, has there been manifested a wider divergence of opinion. The story of ulcer surgery is not, in point of time, a long one; but in its brief course, several significant changes in point of view have occurred.

It may be of interest to review a part of this story, considering these changes, as illustrated by the experience of one hospital—the Roosevelt. As a result of clinical experience through the years, how do our policies and procedures in relation to peptic ulcer differ now from those of an earlier day? What has brought about these changes in our attitude toward this problem?

It is not presumed to attempt here, by the presentation of a great amount of clinical material, nor of a mass of statistical data, to prove the validity of any particular therapeutic method, nor even to justify, on such a basis, the position in which we now stand. The purpose of this paper is merely to record the events of a clinical experience which we believe to be fairly typical, and which may serve to indicate the trend and progress of peptic ulcer therapy.

Major surgery of the stomach is so much an accepted matter of usage today that it is a little difficult to realize how short a distance we are from its beginnings. In Ashurst's International Encyclopedia of Surgery, published in 1884, Henry A. Morris[1] had this to say concerning it: "Gastro-enterostomy is an operation which has been proposed for those cases of cancer of the pylorus in which excision is not possible. It was performed for the first time by Dr. Anton Wölfler, in Vienna, and has since been repeated by Billroth. This mode of treatment appears to have been devised on the spur of the moment, after an exploratory incision had been made in the abdomen of a man who was suffering from cancer of the pylorus, and in whom the operation for removal of the tumor proved to be impossible." Report of this case was published in 1881. Partial gastrectomy, or pylorectomy, was first performed by Péan of Paris, in 1874, but his patient died on the fifth day. He continues: "The first successful results were obtained by Billroth and Wölfler. Though it has now been performed 30 times or more, the operation still has about it so much of the nature of an experiment—perhaps even of a mere surgical exercise—that it is not possible to describe it as a thoroughly recognized surgical

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proceeding." It has been proposed, he said, for cancer and cicatricial stenosis of the pylorus "with most disastrous consequences." "Twice at least, and successfully, it has been done for simple chronic gastric ulcer; and once, also successfully, for stricture of the pylorus following penetrating ulcer."

What advances occurred during the succeeding 20 years in technic, and hence in surgical boldness in attacking the problem of peptic ulcer, may be gathered from a paper by Brewer,[2] in 1907, entitled: "A Critical Review of a Recent Series of Operations Upon the Stomach." In it, he reported 17 cases of suspected peptic ulcer operated upon by him at the Roosevelt Hospital. Of these, 12 showed anatomic lesions while five did not, the diagnosis having been made purely upon clinical observation. He deplored the lack of a certain method of diagnosis, and propounded, as a result of his experience, the following indications for the treatment of benign lesions of the stomach:

(1) Intelligent medical treatment in all primary cases of simple round ulcer. If unrelieved after six weeks of this treatment, operation should be advised.
(2) Operation in all cases of indurated chronic ulcer, and in all cases of recurrent symptoms after primary cure.
(3) Operation in all cases of pyloric stenosis, excepting those due to gummatous infiltration.

Rodman,[3] in 1914, drew a sharp distinction between the treatment of gastric and of duodenal ulcerations. He had advocated, since 1900, the employment of pylorectomy and partial gastrectomy or excision of the ulcer bearing area in the treatment of gastric ulcer, especially in the pyloric region. He condemned the use of gastro-enterostomy alone in the treatment of these lesions, feeling that it failed to protect against the dangers of carcinoma, hemorrhage, and perforation. He had collected and published, at the same time, reports of 205 pylorectomies, with 18 deaths, a mortality of 8.7 per cent. Of local ulcer excisions, there were 171, with three deaths, or 1.75 per cent.

During the years 1912–1914, the writer assisted at an interesting and significant series of observations then being conducted by Brewer and Cole.[4] The question raised was: "Is there reason to believe from our present experience that the roentgen ray will eventually prove as valuable for the diagnosis of surgical lesions of the stomach and duodenum as for the diagnosis of fractures and urinary calculi?" The lack of a certain diagnostic procedure, which he had deplored in his earlier paper, had continued to trouble Brewer, and in characteristic fashion he had proceeded to do something about it. The results seemed to give an affirmative answer to the question. While the first roentgenograms of the stomach employing contrast medium were reported by Hemmeter, in 1896, Cole now added the principle of serial pictures, which greatly increased the value and accuracy of the studies.

Brewer and Cole reported 27 cases, which had been studied and operated upon. In 22 cases, in which a definite diagnosis was made roentgenologically, 20 showed the diagnosis to have been correct. Of five cases in which an

opinion only was expressed, based upon the roentgenologic findings, four showed the opinion to have been correct. The gross result was 89 per cent accurate diagnoses.

This striking advance in diagnostic accuracy was not hailed with complete enthusiasm in all quarters. There was some difference of opinion as to its value. Bevan, for instance, in discussion of the paper, said of the roentgenologic method of diagnosis: "It is of very little value in duodenal ulcer; in not one case out of ten is it of value."

It is certain, in any event, that the diagnostic aid offered by roentgenologic examination materially increased, in our institution at least, the activity of stomach surgery. In 1915, Peck[5] reported 134 operations upon the stomach for benign lesions, performed at the Roosevelt between 1910 and 1915. They were for: Chronic duodenal ulcer, 74; chronic gastric ulcer, 24; chronic gastric ulcer with hour-glass deformity, six; perforated duodenal ulcer, 17 (a surprisingly low figure, comparatively); and perforated gastric ulcer, 13. He observed that modern surgeons of our time have "made safe and a matter of routine operations which a few brief years ago were hazardous in the extreme and uncertain of outcome." He pointed out that at the Roosevelt Hospital gastro-enterostomy had been the routine treatment of chronic ulcer of the duodenum, and that pyloric exclusion or excision of the ulcer had been rarely resorted to. Gastro-enterostomy had, in fact, been performed in 72 of the 74 cases of duodenal ulcer (one pylorectomy and one exclusion accounted for the other two). There had been six deaths, or 8.1 per cent mortality. He reported 58 of these cases traced subsequently. Of these, 51 had remained cured, five improved, and two were unimproved. Thus, he estimated 68.9 per cent of all operated upon as cured, and 88 per cent of those traced. He encountered no case of gastrojejunal ulceration.

In accord with Rodman and earlier authors, he preferred excision in the treatment of gastric ulcers. Of the 30 he reported, 13 received gastro-enterostomies, and the remaining 17 had partial gastrectomies or local excision or cautery puncture in addition. Of the 13 simple gastro-enterostomies, two died after operation, two "subsequently," one developed carcinoma, one was unimproved, and seven were well seven months to five years afterwards. He concluded that for gastric ulcers at or near the pylorus, a pylorectomy was best, while for those situated on the lesser curvature in the pyloric or middle third areas, local excision, preferably with gastro-enterostomy, was indicated. A good gastro-enterostomy stoma, he believed, would continue to function in spite of a patent pylorus, and a functioning stoma would usually be accompanied by a clinical cure.

The succeeding ten years marked a period of great activity in the surgical treatment of gastric and duodenal ulcer at the Roosevelt Hospital. With the benefit of quite accurate roentgenologic diagnosis, an improved operative technic and the assurance gained from the observation of satisfactory results, an increasing number of cases were submitted to operation. Over a period of ten years, from 1917–1927, an average of 50 cases a year were operated

upon. The great majority of these were cases of duodenal ulcer, upon which 412 gastro-enterostomies were performed. It was the exception, during this period, that such cases received preliminary medical treatment, at least in the hospital. Customarily, the diagnosis having been established by clinical findings and roentgenologic examinations, operation was performed rather promptly. The feeling prevailed, at least among the surgeons, that peptic ulcer was essentially a surgical disease and that the surgical treatment accorded it was in the main satisfactory.

It is undoubtedly true that among medical men and gastro-enterologists, at least, this attitude was seriously questioned. Among others, it will be remembered, Bastedo, in this city, valiantly upheld the claims of medical treatment. The literature of the time reflects the controversy which raged between the proponents of the two types of treatment in all parts of the country. As time elapsed, it became increasingly evident that the claims for medical treatment in peptic ulcer, supported by a growing mass of reported results, were not to be denied.

Peck[6] recognized this clearly, and stated, in 1924, that: "We agree with the internists that early, uncomplicated cases (of duodenal ulcer) should first receive medical treatment and that a considerable number of patients are cured, or at least kept in reasonable comfort for long periods of time. We prefer that patients should have had a thorough and intelligent trial of medical treatment before surgery is considered."

In this paper, he brought up to date his report on the results of his ulcer surgery at the Roosevelt Hospital. Since his previous report on 74 cases, in 1914, he had operated upon 122 additional cases of chronic duodenal ulcer, or 196 in all. Gastro-enterostomy had been performed in 191 cases, pylorectomy in three, pyloric exclusion in one, and simple excision in one. Sixteen deaths occurred in this group, a mortality of 8 per cent. As to late results, one case was reoperated upon for gastrojejunal ulcer, and one, after eight years, for a "rigid stoma." Four patients had recurrence of pain and indigestion.

As a result of this continued experience, he stipulated his choice of procedure in various types of duodenal ulcer: (1) Small, single, anterior wall ulcers without narrowing of the gut might have local excision without gastro-enterostomy. (2) Chronic, indurated ulcers, without obstruction, including the chronic perforating variety, might be treated by gastro-enterostomy which, alone, would cure a large percentage. In the more severe cases, a two-stage operation might be employed (gastro-enterostomy, followed by resection 10 to 14 days later) as a safer and easier method than primary resection. (3) For cases with duodenal stricture or so-called pyloric stenosis, gastro-enterostomy was ideal. (4) For cases in which severe hemorrhage had occurred, a gastro-enterostomy plus excision of the ulcer area was advocated.

In these recommendations, a trend toward a slightly more radical attitude begins to be apparent, though Peck was by no means ready to accept the teachings of some of his contemporaries. In fact, in this paper he said: "We have

been considerably disturbed by the tenor of several papers and discussions, and the attitude of a number of prominent surgeons, as expressed at recent important meetings, notably in the New York Surgical Society, during the past year. . . . The attitude which disturbed us was the tendency to advocate radical measures of resection, often of large portions of the healthy stomach, for the surgical cure of this disease." "Our own experience and belief is," he continued, "that simple gastro-enterostomy, properly performed, is curative and adequate in the great majority of chronic duodenal ulcers: That from 80 to 90 per cent of the patients so treated are completely relieved of their symptoms and remain well as they are followed year after year." Further: "We do not believe that gastro-enterostomy should go into the discard in the treatment of duodenal ulcer, nor do we believe that extensive resections of normal stomach are justifiable for this lesion." And again: "We are unconvinced that resection to prevent the formation of gastrojejunal ulcer is a proper procedure in the 98 cases who do not need it to possibly avoid its occurrence in the other two."

His attitude in the matter of gastric ulcer was quite different as, in fact, had been that of almost all surgeons since Rodman's earlier observations. In this same paper, Peck reported on 72 cases of gastric ulcer operated upon since 1910. He did not analyze these cases in detail, but expressed his opinion concerning their appropriate treatment: "Eradication of the ulcer should be a part of whatever operative procedure is adopted," he said. He advocated usually a local excision of the ulcer or its cauterization with a gastro-enterostomy added. Gastro-enterostomy alone, he felt, would fail to effect a cure in many cases, and apprehended the risk of malignancy. In many instances, however, where excision might prove too severe for the patient, a gastro-enterostomy would be palliative, sometimes curative. In ulcers near the pylorus, he felt that partial gastrectomy was usually the best procedure.

I have dwelt at some length upon this paper of Peck's, not only because it represents the prevailing attitude at that time, but also because it expresses the philosophy upon which the treatment of peptic ulcer at the Roosevelt Hospital was then based, and upon which our method and teaching were predicated for many years. With this point of departure, accepting this point of view as fixed, let us carry forward the record of ulcer treatment at the Roosevelt Hospital, bringing Peck's report up to date.

Since Peck's report, that is from 1924 through 1937, there have been operated upon at the Roosevelt Hospital 262 cases (269 operations) for chronic ulcers of the stomach and duodenum, exclusive of operations for acute perforations of primary ulcers. It is of interest to observe that year by year the number of operations performed for these conditions has gradually diminished, and this without a significant diminution of the number of cases admitted. In 1926, for instance, there were 46 such operations; in 1936, but 12. This change has been due primarily to the increasing recognition of the fact on the part of the surgeons that careful medical management is capable of relieving or controlling a very considerable number of these cases. Sandweiss[7]

has recently pointed out, following a careful study of 291 cases, that 90 per cent were rendered symptom-free after a diet-alkali-rest regimen, and that 60 per cent of those resisting this form of treatment responded to some other type of nonsurgical management (injection treatment, release of "tensional states"). We have, as the result of experience and observation, come to the same conclusion as Rankin[8] who says that: "The surgical treatment of peptic ulcer is the treatment of its complications"—obstruction, perforation, repeated hemorrhage, intractability, questionable malignant degeneration. We feel, as does he, that surgery is indicated only in cases where the medical regimen has failed, or where the above complications have resulted.

In fact, it is our practice now to consider all cases of chronic peptic ulcer as medical problems, primarily, and to admit them directly to the medical wards. In this, we are but carrying to its logical end the principle enunciated by Brewer, in 1907, and reaffirmed by Peck, in 1924. In the development of this policy, a considerable change has gradually taken place in our census of ulcer patients. While in the period from 1914–1924, the average admission of chronic ulcer cases to the surgical wards was 39 per year, and to the medical wards 11 per year, in 1931 the medical wards were receiving 60 per year and the surgical 26. This change has been fully justified by the results obtained in simple primary duodenal and gastric ulcer by medical treatment. The records of the medical service show that 170 of 192 cases, or 88 per cent, of primary peptic ulcer admitted during the last five years were discharged symptom-free. We are in substantial agreement with Ochsner,[9] when he says: "It is our firm conviction that there is no surgical treatment of peptic ulcer, and that surgery is indicated only when there are complications."

Not only is no patient now operated upon at the Roosevelt Hospital for chronic peptic ulcer without having had a thorough, controlled, and efficient course of medical management, but each case for whom operation is proposed must be passed upon by a "court" consisting of physician, surgeon, gastroenterologist, and roentgenologist. By this method of conservatism, the patient is given the full benefit of a thorough survey of his problem. In these conferences, we find ourselves guided to our conclusion as to the appropriateness of operation first by the patient's age and a consideration of his general condition; next, by the diagnosis of his lesion, determined as accurately as possible by a combined study of his history, physical and laboratory findings, and roentgenologic examination; next, by a careful evaluation of the rationale and thoroughness of his previous course of treatment, and finally, the generally accepted criteria for the necessity of operation, so well expressed by Lahey,[10] are applied: (1) The persistence of pain in spite of a good trial of adequate medical management. (2) Persistent or recurrent pyloric obstruction. (3) Massive, recurrent hemorrhage in which fatality is threatened. (4) When recurrent hemorrhage occurs, even if symptoms are controlled by treatment. (5) When a lesion of the stomach proves intractable to adequate treatment or suggests the presence of malignancy. In applying these five criteria, we give considerable weight to the changes in appearance of the lesion

by roentgenologic study, as supplementing our interpretation of clinical response.

This plan, as now practiced, has been of gradual development. In studying our operated cases since 1924, it is impossible to fix a date upon which this plan became effective in controlling our procedures. These, it will be seen, have undergone a slow but steady change. Due, in large measure, to the application of these principles, there has been a gradual decline in our utilization of the less radical surgical measures. The intractable type of cases accepted for operation have, in other words, demanded, for the most part, more radical surgery (as Peck himself recognized in his paper in 1924) than was previously accorded to the simple ulcer cases when more were being operated upon. Thus, for instance, while from 1924–1937, 148 gastro-enterostomies were performed, they show, year by year, a gradual diminution. The record for ten years of this operation for duodenal ulcer was as follows: 1926, 27; 1928, ten; 1930, six; 1932, three; 1934, two. The resection type of operation (Polya and Billroth II) for this lesion has shown, on the other hand, a gradual increase. For example: 1927, one; 1929, two; 1935, three; 1936, four.

A study of the entire group of operations during this period may be interesting for comparison with the earlier group, as indicating certain other trends in our approach to the problem. Table I shows the number and results of operations upon cases of chronic duodenal ulcer. Gastro-enterostomy, it will be seen, far outnumbers the other operations. Its good results compare fairly well with Peck's earlier series, its bad results (16 per cent) are nearly the same, and its mortality (6 per cent) a little less. The late results, in all instances, are determined upon the basis of at least a year of postoperative observation; some run as long as nine years.

TABLE I

CHRONIC DUODENAL ULCER

Operation	Number	Good Results		Poor Results	Died	Percentage
		Early	Late			
Gastro-enterostomy	148	45	71	24	9	6
Pyloric operations (Horsley, Finney, Judd)	21	5	12	4	0	
Polya	10	3	7	0	0	
Billroth I	1			1		

Table II analyzes the poor late results of these operations, and indicates the causes of deaths in each, and the early postoperative surgical complications encountered. The single case of Billroth I proved disappointing, developing obstruction at the stoma one year later and requiring gastro-enterostomy, as did one Horsley procedure six months after its performance. It is reasonable to suppose that these late difficulties may have been due to technical imperfections in the original procedures. With the one exception noted, the pyloroplastic operations gave good results and were unattended by either late or early complications as far as they have been followed. The Polya opera-

tions, though few, had no mortality, and as yet no complications, though three of the ten have not been traced.

TABLE II

CHRONIC DUODENAL ULCER

	Gastro-enterostomy (148)	Pyloro-plasty (20)	Polya (10)	Billroth I (1)
Early Surgical Complications (Survived) ..	(11)			
Hemorrhage (postoperative)	1			
Obstruction (+ entero-enterostomy)	7			
Abscess	1			
Evisceration	2			
Causes of Death	(9)			
Peritonitis	3			
Embolism	1			
Shock	1			
Cardiac	1			
Obstruction (+ entero-enterostomy)	2			
Evisceration	1			
Poor Late Results	(24)	(4)		(1)
Pain, vomiting, or both	16	3		
Recurrence of ulcer	4			
Recurrent hemorrhages	2			
Marginal ulcer	1			
Gastrocolic fistula	1			
Obstruction		1		1

Although they play no part in this study, it may be interesting to note in passing that eight cases of acute perforation of duodenal ulcer received a gastro-enterostomy in addition to ulcer closure. Three of these died, a mortality of 37 per cent, and we have abandoned the procedure except as a matter of inescapable necessity.

The attitude toward gastric ulcer, even from the earliest days, has been marked by a desire on the part of the surgeon to rid his patient of a menacing pathology. Whether one believes, with Lahey,[10] that cancer does not supervene in more than 5 to 6 per cent of gastric ulcer, or with Hinton,[11] who expresses doubt whether this ever occurs at all, or whether one is uncertain on the question and takes into consideration only the dangers of hemorrhage and perforation, one must agree with most surgeons that the unhealing, intractable stomach ulcer is better out than in. As in the cases of duodenal ulcer in this series, most of the gastro-enterostomies, with or without ulcer excision, were performed in the earlier years. But two gastro-enterostomies have been performed for chronic gastric ulcer since 1931. The Polya operations are scattered pretty well throughout the series in point of time, but distinctly outnumber the gastro-enterostomies during more recent years.

The results of these operations for ulcer of the stomach are shown in Tables III and IV. The relatively high mortality among the simple gastro-

enterostomy cases is in some degree attributable to the fact that this operation was more likely to be performed, as a less severe procedure, on the more debilitated patients. Undoubtedly, though, whatever the operation, the gastric ulcer patient appears to be a poorer risk from the surgeon's point of view. Gastro-enterostomy with local ulcer excision or cauterization, perhaps because applicable to the smaller and simpler ulcerations, gave the best early results in this group.

TABLE III

CHRONIC GASTRIC ULCER

Operation	Number	Good Results		Poor Results	Died	Percentage
		Early	Late			
Gastro-enterostomy	17	4	5	3	5	30
Gastro-enterostomy + local excision or cauterization	15	6	7	2	0	
Polya	21	5	12	1	3	14
Billroth I	2		2		0	
Billroth II	1		1		0	
Sleeve resection	1		1		0	

TABLE IV

CHRONIC GASTRIC ULCER

	Gastro-enterostomy (17)	Gastro-enterostomy + Excision or Cautery (15)	Polya (21)
Early Complications (Survived)			
Obstruction		1	1
Hemorrhage	2		
Causes of Death	(5)	(0)	(3)
Cardiac	1		
Shock	1		
Evisceration	2		
Uremia	1		
Peritonitis			2
Pneumonia			1
Poor Late Results	(3)	(2)	(1)
Pain	2	1	1
Developed carcinoma	1		
Recurrence of ulcer		1	

These tables, representing the treatment accorded to primary ulcerations of the stomach and duodenum, do not by any means tell the whole story. There is another group of cases representing late results and complications of previous ulcer surgery that is very significant. These cases, shown in Table V, have been the most difficult to deal with technically and have often presented problems of some complexity. They are interesting as showing what may, and frequently does happen to the patient upon whom some surgeon has previously exercised his best judgment and skill. It is the occurrence of these cases that it should be our particular aim

TABLE V

OPERATIONS FOR OTHER THAN PRIMARY ULCER

Late Complication of:	Complications					Treatment						Results		
	Recurrence of Ulcer	Hemorrhage	Marginal Ulcer	Gastrocolic Fistula	Obstruction of Stoma	Billroth II	Polya	Gastroenterostomy	Horsley	Take-down of Gastroenterostomy	Suture of Perforation	Good	Poor	Died
Gastro-enterostomy (14 cases)	2	2	7*	2	1	4	5	1‡		2	2	8	5	1§
Suture of perforation—gastric (2 cases)	2							2				1	1	
Suture of perforation—duodenal (2 cases)	2						1		1			2		
Pyloroplasty (3 cases)	1		1†		1		1	1			1	3		
Billroth I (2 cases)	1				1			2				1	1	
Polya (1 case)			1†								1		1	

* Two acute perforations.
† Acute perforation.
‡ Anterior, plus entero-enterostomy.
§ Gastrocolic fistula, peritonitis.

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to prevent. The cases in this group have come to us from various sources; some from our own services. It is of interest to note that, with the exception of one Polya resection that suffered an acute perforation of a gastrojejunal ulcer at the end of six months, all the other complications occurred from one to 18 years after the original operation. Of the 14 complications following gastro-enterostomy, five occurred more than ten years after the primary operation. It is obviously unfair to evaluate the results of any of these gastric operations upon the basis of "five-year cures," or in fact upon any arbitrary time standard. Neither is it fair, since in any series so many cases are lost track of in the course of time, to consider all cases as cured who fail to return even after several years of follow-up.

It is, among other things, this difficulty in evaluating late-results that has led to such differences of opinion as to the proper choice of operation for chronic peptic ulcer. To gain some idea of the scope of this controversy, a consideration of Fogelson's[12] exhaustive review of recent literature is recommended. The comments of authorities on both sides of the question of radical surgery as opposed to conservative operations are impressive, but confusing. The present contribution, with its small number of cases, cannot hope to add much to what has been said and written by so many. Yet the experience that these cases represent has been important to us, and has led to a certain viewpoint which may be briefly expressed.

As to chronic duodenal ulcer, we now feel that once the resources of medical treatment have been exhausted, without lasting benefit to the patient, he should be operated upon. This naturally brings to surgery a group of cases suffering from scarred or sclerosing ulcers, ulcers penetrating the head of the pancreas, ulcers invading blood vessels (usually in the pancreas), and ulcers which have caused duodenal or pyloric stenosis. For such ulcers, especially in the younger age group with high gastric acidity, we believe that extirpation of the ulcer and removal of the most actively acid-secreting portion of the stomach is best. In this, we find ourselves in agreement with Balfour,[13] who feels that in such cases, where motor and secretory activity are marked, the recurrence rate of ulcer is likely to be high. While resection, of the Billroth II or Polya type, he finds, may result in subsequent jejunal ulcer, this is less likely to occur than in gastro-enterostomy, and recommends, as do Ochsner[9] and Engel[14] that such a resection should be performed quite high in the stomach. Page and Rankin[15] continue to support gastro-enterostomy as satisfactorily altering the mechanism and chemistry of the stomach. Snell[16] claims a 30 to 50 per cent reduction of acidity following this operation. Engel[14] feels that the effects on neutralization and on pepsin action by this means are greatly overrated. Ochsner[9] believes that the employment of a gastro-enterostomy in such actively secreting stomachs, subjecting a susceptible part of the intestine to the action of acid chyme, is an unwise procedure, and that it favors the development of marginal or jejunal ulcers.

While we have insufficient data in our own series to indicate the late results of our own resections, we have been impressed by the satisfactory early

recovery of those patients, and by the fact that the mortality has been low, even as compared with gastro-enterostomy (no death in ten cases of resection, 6 per cent in 148 patients with gastro-enterostomy). The late, unfortunate complications of previous gastro-enterostomies seen in this series have no doubt influenced us as well, and have led to the feeling that, in this particular type of case, resection gives the better hope of ultimate success. The first surgical operation performed upon a patient presents the best opportunity for securing a lasting good result. The technical difficulties and dangers of secondary operations are too great for comfort.

In the class of patients with very high acidity, we believe that partial resections of the stomach have certain advantages over the pyloroplastic or gastro-duodenal anastomosis type of operation (Finney, Judd, Horsley, Billroth I). There is considerable question, as voiced by Balfour,[13] whether these operations in the pyloric area adequately control the factors precipitating ulceration. They are of distinct value, however, in cases of long standing pyloric narrowing, where, as in older patients particularly, the acid secreting activity of the stomach is appreciably diminished and where prolonged distention of the stomach with stenosis and gastritis has produced a certain atrophy of the mucosa. They have the distinct advantage, too, in older patients, of being primarily safer than the more radical procedures. The same may be said as regards the applicability of gastro-enterostomy. We would reserve this operation, in duodenal ulcer, for cases of stenosis with subnormal gastric acid values. In these, its results have been most satisfactory.

In the performance of resections for duodenal ulcer, it is ideal to remove the ulcerated area at the same time. This, however, owing to marked involvement of the pancreas or to a somewhat debilitated condition of the patient (as in bleeding ulcer, for instance), may not always be expedient, as it may involve too grave a risk. Then, as Lahey[10] recommends, the ulcer may be left *in situ*, though now protected from further chyme irritation, with very good prospect of a satisfactory result. Occasionally, recurrence will be seen, but this must be weighed against the risk involved in resection for the particular case. In the cases of repeated, bleeding duodenal ulcer, it has been our practice not to interfere surgically until the hemorrhage has been controlled and the patient restored, as far as possible, to good condition. The mortality from primary hemorrhage, we believe, is not so great as would be the case were surgery employed in the instance of dangerous, apparently uncontrolled, bleeding. Even though we are performing more resections than gastro-enterostomies, our views on the surgical treatment of chronic duodenal ulcer still agree definitely with those of the less radical school. Rankin,[8] one of the chief proponents of conservative stomach surgery, says, as did Peck, that the cases of recurrent hemorrhage, of ulcers penetrating the head of the pancreas, and of ulcers which are intractable owing to induration, infiltration, and presence of excessive callous, merit resection. With the method of case selection we now employ, and operating as we do, in consequence, on fewer patients, we find that such cases as he considers proper subjects for resection far exceed the

10 per cent of the whole surgical group which is his estimate of their relative occurrence.

In the field of chronic gastric ulcers, there is, as has been indicated, but little question as to the desirability of the extirpation of the lesion. Here, the same criteria as to operability are recognized as in the case of the duodenal lesion, but always with the question in the surgeon's mind as to the possibility of malignancy. It is this question, particularly, that leads to the more radical viewpoint in the handling of these lesions. While the local excision or cautery puncture of the smaller ulcer, together with gastro-enterostomy, may serve to secure and maintain a cure in many cases, we are inclined to favor resection, where the risk is not too great for the patient, for two reasons: First, by removing the vulnerable area of the stomach and by altering more profoundly the stomach chemistry, it makes recurrence of ulceration less likely. Second, it distinctly diminishes the probability of advancing malignancy should there be any suspicion of cancer. Balfour[13] reports 6 per cent of cancers developing after gastro-enterostomy in cases where the ulcer was not suitable for removal. It has been amply demonstrated that it is by no means always possible to differentiate cancer from ulcer at operation, and even in the laboratory this distinction sometimes presents difficulties. Again, however, the risk to the individual patient must be carefully weighed, and the decision as to type of operation determined primarily on that basis. It will be recalled that, in the present small series of gastric ulcers, we had five deaths after simple gastro-enterostomy in 17 cases, three deaths in 21 Polyas, and none in 15 gastro-enterostomies with local excision, but one of the latter group (not included in the table because review of the original sections showed the pathology) died later of cancer of the stomach.

The large ulcers, associated, as they often are, with considerable areas of surrounding gastritis and frequently with multiple ulcerations, do not lend themselves well to local excisions. Not only are such excisions and sleeve resections open to the same objections as is the excision of smaller ulcers, but they present, in addition, fully as great technical difficulty and risk as a well-planned resection. Moreover, they are likely to leave a deformed and often physiologically unsatisfactory stomach. In these large ulcers, therefore, resection is preferred where the patient's condition will permit. On this matter of choice of operation, Balfour[13] says: "When partial gastrectomy can be performed with little more risk than excision and gastro-enterostomy, it can never be condemned for gastric ulcer." Hinton[11] thinks the operative mortality is too high to warrant resection as a protection against cancer.

In the difficult and complex problems presented by the patient who suffers from recurrence of duodenal ulcer, from jejunal or marginal ulcer, or from stenosis of the stoma following a lesser type of operation, resection remains usually the only recourse. These secondary operations involve considerable technical difficulty and are accompanied by a definitely increased hazard. While it must be recognized that the mere taking down of a gastro-enterostomy, or the adding of a fresh one to relieve obstruction is by no means certain

to result in permanent benefit, the patient's individual ability to withstand a proposed resection must be taken seriously into account in choosing the operation to be performed, and may lead one to choose the lesser alternative.

The question of the patient's condition and ability to withstand the operation that may be planned for him is of paramount importance. The success of these various operative procedures will depend upon the judgment with which we apply them, not to the lesion, but to the patient. In this regard, the prolonged period of medical care that precedes operation has proven of great help to us in gaining, from the patient's physician, a clear idea of his physical status. The "court" that sits upon his case brings further judgment to bear upon this point, and tends to curb any undue individual enthusiasm for some particular operative method. But whatever program is laid down for the operation must be subject to variation, modification, or abandonment by the surgeon if the conditions found at operation warrant. An inflexible attitude of adherence to a set plan is never more to be deplored than when applied to a problem of gastric surgery.

A further safeguard for the patient lies in his preparation for the operation. It is our plan, when all that can be done for him in the way of elimination of foci of infection and rectification of constitutional defects has been accomplished during his medical regimen, to give the patient a rather intensive preoperative preparation. Where no contraindication exists, his stomach is lavaged once or twice daily for several days to relieve inflammation, restore tone, and overcome retention if it exists. During the three or four days preceding operation, he is given daily two infusions of 1,000 cc. of glucose, 5 per cent, in normal saline, to build up a good fluid and glycogen reserve. If his blood count is subnormal, he receives one or more transfusions before operation. As a regular measure of postoperative care, he receives a transfusion upon leaving the table, and during the succeeding days, until he takes his fluids in adequate quantity by mouth, his water balance is kept up with sufficient infusions for the purpose, supplemented by tap water by rectum. Once recovered, experience has amply demonstrated the necessity for prolonged, perhaps permanent, observation and control of these patients with respect to their habits, activity, and diet.

We are encouraged in the pursuit of the policies and practices above outlined by the early results in the present group of cases. The 14 year period covered by this report shows a mortality in Polya and Billroth II resections, for all types of primary peptic ulcer, of 9.3 per cent. This compares favorably with the 8.5 per cent mortality of gastro-enterostomies during the same period, when one considers the 16 per cent of recorded late poor results with gastro-enterostomy, as against the 3 per cent of poor results with resection. These figures, too, do not take into account the cases of secondary complications shown in Table V.

We are yet far from content with the present status of ulcer therapy. We recognize, as Ochsner[9] enumerates them, the precipitating factors which lead to ulcer formation or promote its recurrence; namely, hypersecretion, hyper-

acidity, focal infections, reflex pylorospasm, and gastric trauma. In the control of these, we have progressed a little way. But the underlying factors, the constitutional, predisposing, individual causes of susceptibility to this disease still elude us. The search for the true etiology of peptic ulcer must continue to be pursued vigorously. Whether we shall find the answer in the field of the autonomic nervous system, or of the endocrine glands; in the manifestations of toxicity or of infection, we are, as Adams[17] points out in reviewing the various hypotheses, still in the dark. For the present, we must continue to treat the complications of this disease according to our best surgical judgment, but when the true key is found, the surgeon may thankfully lay down his scalpel and relegate the control of peptic ulcer to the competent hands of his medical confreres.

SUMMARY

(1) Our experience at the Roosevelt Hospital is recorded to show how we have come to consider chronic peptic ulcer as essentially a disease for medical treatment, and how we have reached the belief that the surgical treatment of ulcer is essentially the treatment of its complications, rather than of the disease itself.

(2) We are treating to-day, by surgical intervention, far fewer cases of gastric and duodenal ulcer than was formerly our practice.

(3) Before being submitted to surgery, all cases must have had the benefit of a carefully controlled medical management.

(4) The decision to employ surgery in a given case rests with a "court" consisting of physician, surgeon, gastro-enterologist, and roentgenologist.

(5) The application of these principles has resulted in bringing to surgery a relatively larger number of patients in whom the more radical types of operation are required.

(6) The type of operation to be employed in each case is determined by certain guiding principles:

(*a*) It must be of such a nature that the particular patient can tolerate and survive it.

(*b*) It should aim not only at alleviation of symptoms, but should give freedom from likelihood of recurrence or of complications, both early and late.

(*c*) The ideal procedure having been determined, it should be abandoned or modified if the condition found at operation warrants.

(7) Careful preoperative preparation of each patient is required.

(8) Careful postoperative management is essential. The use of transfusions after stomach operations has become virtually a matter of routine.

(9) Careful late postoperative management is essential. The factors believed to have been involved in the original production of the ulcer must continue to be eliminated. For this purpose, a consistent and prolonged follow-up is requisite.

(10) A comparison of mortality and results encourages us in the continued employment of the practices outlined.

(11) We continue, hopefully, to seek the true etiology of ulcerations of the stomach and duodenum in order that our treatment of them may become less symptomatic and more rational.

(12) We believe that future success in the treatment of peptic ulcer lies in the direction of its prevention or early control, through a better understanding of its causes and nature, rather than in that of more brilliant, daring, and technically improved surgical attack upon the stomach and duodenum.

REFERENCES

[1] Morris, Henry A.: In Ashurst's International Encyclopedia of Surgery, **5**, 1111–1112, 1884.

[2] Brewer, George E.: A Critical Review of a Recent Series of Operations Upon the Stomach. ANNALS OF SURGERY, **45**, 687–698, May, 1907.

[3] Rodman, Wm. L.: Pylorectomy and Partial Gastrectomy, or Excision of the Ulcer-bearing Area in the Treatment of Gastric Ulcer. Trans. Amer. Surg. Assn., **32**, 145–163, 1914.

[4] Brewer, George E., and Cole, Lewis G.: The Roentgenologic Diagnosis of Surgical Lesions of the Stomach and Duodenum. Trans. Amer. Surg. Assn., **32**, 108–144, 1914.

[5] Peck, Chas. H.: Benign Lesions of the Stomach and Duodenum. J.A.M.A., **65**, 659–663, August 21, 1915.

[6] Peck, Chas. H.: Present Status of Surgical Treatment of Chronic Duodenal and Gastric Ulcer. ANNALS OF SURGERY, **80**, 31–44, July, 1924.

[7] Sandweiss, D. J.: Comparative Results with Dietetic, Parenteral, and Surgical Treatment of Peptic Ulcer. J.A.M.A., **108**, 700–707, February 27, 1937.

[8] Rankin, F. W.: Present-day Management of Peptic Ulcer. Kentucky Med. Jour., **35**, 209–214, April, 1937.

[9] Ochsner, A.: Rational Treatment of Peptic Ulcer According to Physiologic Principles. Texas State Jour. Med., **32**, 645–649, February, 1937.

[10] Lahey, Frank H.: Indications for Surgery in Peptic Ulcer. Surg. Clin. N. Amer., **17**, 621–627 and 687–692, June, 1937.

[11] Hinton, J. W., and Trubeck, M.: Transformation of Gastric Ulcer into Gastric Carcinoma. Surg., Gynec., and Obstet., **64**, 16–21, January, 1937.

[12] Fogelson, Samuel J.: Collective Review Gastroduodenal Ulcerative Disease. Surg., Gynec., and Obstet., **65**, 1–16 of International Abstract, July, 1937.

[13] Balfour, Donald C.: Indications for Operation in Cases of Gastric Disease. Surg. Clin. N. Amer., **17**, 947–960, August, 1937.

[14] Engel, G. C.: Gastro-enterostomy *vs.* Polya Operation in Duodenal Ulcer. Southern Surg., **6**, 231–236, June, 1937.

[15] Page, W., and Rankin, F. W.: Review of Ulcer Surgery at the Presbyterian Hospital (Philadelphia), 1921–1935. Am. Jour. Surg., **37**, 219–231, August, 1937.

[16] Snell, A. M.: Behavior of Stomach After Operation for Duodenal Ulcer. Am. Jour. Surg., **35**, 45–55, January, 1937.

[17] Adams, W. P.: Factors in the Etiology of Peptic Ulcer. Southern Med. and Surg. Jour., **99**, 221–226, May, 1937.

GASTRO-ENTEROSTOMY*

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DURING my early surgical training I was taught that acute ulcers of the stomach and duodenum were medical conditions and did not require operation unless repeated attempts at cure had failed or complications developed which made surgical interference advisable or necessary.

The next step in my surgical education was the teaching that the simplest operation which will relieve the patient's symptoms, cure the condition, and has the lowest mortality, is the operation of choice. As the result of this teaching and my own observations I have for a long time been under the impression that gastro-enterostomy is a very satisfactory procedure if applied in the cases in which it is indicated, and if performed with all the attention to detail which it requires. Consequently I have viewed with misgivings the campaign against this operation and in favor of routine radical procedures.

It has become, and is my feeling today that such teaching is not in the best interest of the patient. No harm will probably be done in the larger surgical centers, because the members of their staffs have sufficient material to make their own observations and form their own opinions. But much harm may be done by inducing surgeons who infrequently have occasion to perform an operation on the stomach to give up a well tried, and on the whole satisfactory, operation which carries with it a low mortality, for a radical procedure with its resultant higher mortality.

In order to determine whether my impressions were erroneous I decided to examine the value of gastro-enterostomy on the basis of my own material as well as that of others. In addition to my personal cases I have, therefore, made a study of the more recent literature.

Gastro-enterostomy was first performed by Wölfler[1] at Billroth's Clinic, in 1881. The patient was a man with an inoperable cancer of the pyloric end of the stomach. Rather than close the abdomen without attempting to give relief, Wölffler decided to make a new opening between the anterior wall of the stomach and a loop of jejunum, and called the operation "gastro-enterostomie." The patient recovered from the operation. Soon thereafter his chief, Prof. Theodor Billroth, performed the same operation on a similar case. This patient succumbed as the result of persistent vomiting. An autopsy showed that spur-formation, due to prolapse of the intestinal wall opposite the stoma, had divided it into two openings, the larger one communicating with the proximal, and only a very small one with the descending loop. Stomach contents would thus regurgitate easily into the ascending loop and distend it until

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the pressure projected it back into the stomach, while almost none passed into the lower intestine. The dangers of anterior gastro-enterostomy by a vicious circle were, therefore, recognized early. During the following years the principles of the operation were applied to nonmalignant pyloric obstructions, notably by Rydigier,[2] who, in 1884, operated upon an emaciated boy, age 20, with an obstruction due to a stenosing ulcer, and obtained a successful result.

In order to try to avoid the difficulties of a vicious circle so frequently following a long loop anterior gastro-enterostomy von Hacker[3] conceived the idea of performing a retrocolic, posterior gastro-enterostomy. With slight modifications or additions this has remained the operation of choice in those cases in which no technical obstacles to its performance are encountered.

It is apparent that the operation was conceived as a side-tracking procedure for the relief of pyloric obstruction, whether malignant or benign. The results of the operation in this type of case were so very gratifying, that the thought suggested itself to apply the same principle to ulcers not associated with mechanical obstruction. It was hoped that by putting the ulcer at rest by an exclusion operation, healing might result. The unexpectedly good results which followed this procedure led to investigations as to the cause of the beneficial effect.

Its action in mechanical obstruction is easy to understand, because there it acts actually as a side-tracking operation and brings relief by drainage. Its action in ulcer cases without obstruction is not so easily explained. If a certain amount of pylorospasm is present as the result of hyperacidity and irritation from a near or more distant ulcer the same explanation may suffice. However, if there is no obstruction at all the pylorus remains patent after gastro-enterostomy and food may, and often does, leave the stomach by the two openings. The opinion then gained ground that the influx of duodenal contents through the new stoma alkalinizes the stomach contents and, thereby, induces a beneficial healing action on the ulcer. However, this question still remains a controversial one. Nevertheless, it is true that ulcer patients have been relieved by the operation in a high percentage of cases and one has to assume that the altered physiology of the stomach in some way exerts a beneficial influence. It is readily conceded that there are a certain percentage of ulcer patients who are not relieved or cured by gastro-enterostomy, or in whom a new group of symptoms due to secondary ulcer formation at or near the stoma is initiated. These two reasons, failure to cure the ulcer and an alleged high frequency of secondary gastrojejunal or jejunal ulcers, have led some surgeons to the conclusion that radical surgery, meaning by that partial or subtotal resection of the stomach, is indicated for the cure of both duodenal and gastric ulcers. Advocates of radical surgery as a routine procedure claim an incidence of 10 to 20, and even more, per cent of jejunal ulcers following simple gastro-enterostomy. This is at variance with the experience of most surgeons, and most of the large clinics of the country, which report only 3 to 4 per cent of such ulcers.

Nevertheless, the campaign in favor of radical surgery has succeeded in

discrediting gastro-enterostomy to such an extent that many surgeons are disinclined to advocate it.

It is, therefore, timely to determine whether it is justified to abandon an operation which, through the years, has given such satisfactory results, for a more radical procedure with its attendant higher mortality, and a doubtful guarantee that it will not be followed by the very sequelae for the avoidance of which it was instituted.

We may quickly pass over the question of gastro-enterostomy for carcinoma of the stomach. It is at most a palliative measure, and is used only in those patients in whom a radical operation cannot be performed because of technical difficulties or because the general condition of the patient does not permit. In such individuals the mortality is, therefore, expected to be high. When technically possible to be performed without undue tension on the suture lines, the results are good, and usually insures the patient relief of symptoms and several months of comfortable life.

The real interest in the operation of gastro-enterostomy centers about its use in cases of gastric and duodenal ulcer, as well as in the complications or sequelae of these conditions, namely, hemorrhage, local perforation and scar contracture with consequent pyloric obstruction.

All surgeons agree that the problems connected with the surgical treatment of gastric ulcer differ considerably from those associated with duodenal ulcer, and that they should, therefore, be considered separately.

In duodenal ulcer a side-tracking operation by gastro-enterostomy is possible and the ulcer may be put completely at rest, whereas, in gastric cases this is usually not the case. Only in those ulcers situated near the pylorus and producing some degree of spasm or stenosis may one reasonably expect such a result. Cautery destruction or excision of ulcers has not been as successful as was hoped for. There is in addition a tendency to cancerous degeneration of gastric ulcers variously estimated at from 10 to 20 per cent. This, coupled with the general experience that conservative treatment in gastric ulcer is less satisfactory than in duodenal ulcer, has induced most surgeons to advocate, and perform, partial gastrectomy in those cases which have not responded to medical treatment.

We fully agree with this view and have for years performed this operation as a method of choice. The fact that the duodenum is not involved, and permits safe closure of the stump, usually makes it less dangerous than a resection for duodenal ulcer. Unless the ulcer has perforated and is extensively adherent to the pancreas, there is no unusual risk connected with the operation in a patient in fair general condition.

While conceding that resection is the treatment of choice, gastro-enterostomy, nevertheless, has a definite, though limited, place in the treatment of gastric ulcer. This is particularly true of the large hypertrophic ulcers of the pylorus and prepyloric region, which not infrequently simulate inoperable carcinoma. It is also of value in ulcers near the pyloric end with inflammatory reaction and extensive adhesions, and in some of the ulcers situated high on

the lesser curvature. Here they are inaccessible, and attempt at removal by resection dangerous. Closure so high up is difficult and fatal leakage may result. Relief of symptoms or cure may be obtained after gastro-enterostomy, and is best explained by subsidence of pylorospasm with better drainage, as well as by alkalinization of the stomach contents.

The question of the surgical treatment of duodenal ulcer is the one we are chiefly interested in at this time because it is this group in which we are asked by the advocates of routine radical operation to substitute gastric resection for gastro-enterostomy.

The original communications on this subject by Haberer,[4] and other German authors, as well as those by its American advocates, stress failure to cure duodenal ulcer by gastro-enterostomy, as well as the frequency of postoperative marginal or jejunal ulcers as reasons for abandoning gastro-enterostomy and substituting therefore partial or subtotal gastrectomy. A. A. Berg[5] bases his plan of treatment on the necessity of removing the three factors which he holds responsible for the formation and development of an ulcer; namely, chronic specific gastritis, the presence of free hydrochloric acid in the stomach, and the presence of a secondary infection in the stomach or duodenum. The procedure that will most nearly accomplish this is partial or subtotal gastrectomy. On the basis of a large material he builds up a very good case in favor of early radical surgery, and describes an operative procedure which, in his hands, has been followed by a very low operative mortality and excellent late results.

Lewisohn,[6] in an analysis of 68 cases of gastro-enterostomy with or without pyloric occlusion, found a perfect result was obtained in 47 per cent, a fair result in 19 per cent, and the remaining 34 per cent had gastrojejunal ulceration, He explains this unusually high incidence on the basis of careful personal reexamination of the patients, and evidently believes other surgeons would find a similarly high incidence if they followed their cases more carefully. He rejects the possibility of faulty technic as a factor, but definitely holds persistent hyperacidity in the gastro-enterostomy cases responsible for the continuation or aggravation of symptoms. It is his firm belief that the important factor in the prevention of subsequent gastrojejunal ulcers is anacidity, not the technic of the operation, and that such an anacidity is best obtained by subtotal gastrectomy. Several other surgeons share this view and are advocating radical procedures as a routine, while another group are performing resections on carefully selected cases only. There is a great deal of experimental evidence to support the attitude of these surgeons, though many reports contradict one another and leave one in somewhat of a state of confusion.

Recent observations by DeBakey[7] on dogs upon which a von Eiselsberg exclusion was performed, in conjunction with an anterior gastro-enterostomy, showed 50 per cent jejunal ulcers after operation. The ulcer always appeared in the efferent loop where the acid neutralizing power of the duodenal contents had been largely lost. He concludes that the jejunal mucosa is func-

tionally unable to receive acid gastric chyme and that inadequate neutralizing power of the duodenal secretion apparently plays an important rôle in ulcer production. In order to prove the protective value of the various components, he diverted the pancreatic juice from the duodenal contents, so that the only protective agents remaining were bile and succus entericus. After such a procedure ulcers developed in 70 per cent of cases. If he also diverted the bile, ulcer developed in 90 per cent of cases, and not only in the efferent loop but also at the anastomotic line and opposite the stoma in the jejunum. He holds bile to be the most significant protective factor. The author further stresses the importance of foreign body such as suture material as a possible cause of ulcer and quotes a number of authors in support of this view. However, he also cites several cases in which suture material was found at the stoma without ulcer formation.

In a more recent paper on the physiology of peptic ulcer, DeBakey emphasizes the fact that no single factor can be held responsible for peptic ulceration in general, which is a viewpoint held by most thoughtful observers.

Against these views of the proponents of routine radical surgery there are any number of articles based upon clinical observation citing excellent results following gastro-enterostomy. There are also numerous laboratory studies which show the beneficial effects of the operation upon the acidity of the stomach.

Heuer,[8] in a recent paper on the choice of operations in the treatment of peptic ulcer, mentions three methods of operative procedure for duodenal ulcer: Local excision of the ulcer combined with some form of pyloroplasty; a simple gastro-enterostomy; and pylorectomy or partial gastric resection. Each type is indicated in certain cases. He collected 1,559 gastro-enterostomies for duodenal ulcer from the recent literature, which showed a general mortality of 6.8 per cent. The late results showed 85 to 92 per cent satisfactory relief from symptoms in American clinics. Gastrojejunal ulcer as a late complication was found in about 3 per cent of cases in the experience of 17 authors from various countries. A study of the results of eight authors showed jejunal ulcer following partial gastrectomy in 1.9 per cent of cases.

Judd and Hazeltine,[9] after a comprehensive review of a large series of excisions for duodenal ulcer, cite two reasons why the operation of excision of ulcer of the duodenum has not gained rapidly in popularity: (1) That the results of gastro-enterostomy when performed for this condition have usually been satisfactory; and (2) unless the first portion of the duodenum is free and mobile, excision of the ulcer may be very difficult. Unless it is possible to excise the ulcer with less risk and better results than attend gastro-enterostomy, there is no occasion for this operation. They conclude that gastro-enterostomy will probably remain the popular operation for duodenal ulcer. It is satisfactory in all cases except in those in which secondary ulcers develop, hemorrhage occurs, or bleeding may continue.

St. John,[10] in a comparison of follow-up statistics between 119 cases of gastro-enterostomy and 76 cases of partial gastrectomy, reports a mortality of

15.1 per cent in the former and 19.6 per cent in the latter. Of the gastro-enterostomy cases there were 92.8 per cent symptom free or improved, and of the gastrectomy cases, 96.8 per cent were symptom free or improved. The unusually high postoperative mortality is due to pulmonary complications. The author reports marginal ulcer in 6.9 per cent of the gastro-enterostomies, and in 3.6 per cent of the resections.

Balfour[11] presents a very complete analysis of 500 cases of gastro-enterostomy for duodenal ulcer which had been observed after a minimum of five years after operation. The operation was performed in most instances in chronic cases. Eighty-seven per cent obtained relief, which they had been unable to obtain by any other means, and in 69 per cent it was so complete that the patient was not conscious of having a stomach. The operative mortality was 1.8 per cent. In no case did perforation of the duodenal ulcer occur after gastro-enterostomy had been performed, which makes him feel that this operation, apparently, affords absolute protection against this serious complication. Likewise in none of the cases of this series did obstruction of the pylorus develop subsequent to operation. However, it seemed more difficult to secure permanent protection against hemorrhage, for 45 of the 500 patients, or 9 per cent, had one or more hemorrhages, but only one died as the result of it. The protection afforded by gastro-enterostomy against the formation of a secondary chronic ulcer is approximately 96 per cent, for in only 3.26 per cent, gastrojejunal or jejunal ulcer occurred in a period of ten years or more after operation.

Balfour also studied 100 cases of gastro-enterostomy performed for gastric ulcer. Although he believes in removing the lesion if reasonably possible, he is convinced that gastro-enterostomy alone is the operation of choice in those cases in which the size of the lesion, its situation, or the age or condition of the patient makes its removal a difficult or hazardous procedure. The operative mortality in this group was 3 per cent. Seventy-nine per cent of the patients, five years or more after operation, were relieved, with complete relief being obtained in 60 per cent. No gastrojejunal or jejunal ulcer developed.

He feels that gastro-enterostomy has its greatest application in cases of chronic duodenal ulcer in which there is impairment of motor function and in which the acid values are not unusually high. Since impaired motor function usually occurs late in the development of duodenal ulcer, it is in the most chronic cases that gastro-enterostomy is so effective. In a more recent paper on peptic ulcer and its surgical treatment by conservative measures, Balfour repeats that he believes gastro-enterostomy to be the most useful of the different procedures which may be employed. He states that if the operation could be confined to the more chronic cases there would be no criticism of it, but the very fact that it did bring about such spectacular results in this group led to its employment in cases in which there was no impairment of motor function and in cases in which symptoms were of short duration. It is probably for this reason that so much criticism of the operation has arisen, because it is in

this group that disappointing results are most likely to occur. He further states that when the indications for gastro-enterostomy are understood and carefully observed, the results are strikingly similar. Between 80 and 90 per cent of patients have obtained satisfactory results following gastro-enterostomy, and there have been only 3 to 4 per cent with subsequent jejunal ulcer formation.

Walters[12] disposes of the arguments in favor of routine radical surgery, particularly with regard to the frequency of gastrojejunal ulcers following gastro-enterostomy, by stating that he has rarely seen them, not more frequently than after gastric resection; and again, relative to the statement that associated gastritis is a cause of duodenal ulcer, as well as of recurrence of symptoms after gastro-enterostomy, by stating that gastritis has rarely been found in the specimens removed by them.

He also quotes from a collective investigation by the British Medical Association, in 1928, into the after-history of gastro-enterostomy cases operated upon during 1920 to 1924. According to this report gastrojejunal ulcer occurred in 2.8 per cent of 744 cases in which gastro-enterostomy was performed for duodenal ulcer. The postoperative mortality was 5 per cent. The results of the operation were satisfactory in about 90 per cent of the cases.

His paper further deals with the question of tissue resistance and tissue susceptibility, particularly to hydrochloric acid, and makes a comparison between the relative achlorhydria obtained in groups of cases treated surgically by different methods. He concludes that tissue resistance and tissue susceptibility to hydrochloric acid, and their measurement, are the factors upon which successful treatment of duodenal ulcer depends regardless of the surgical procedure employed, for, if tissue resistance to hydrochloric acid is satisfactory, ulceration will not recur, whereas, when tissues are susceptible to the hydrochloric acid of the gastric secretion, ulcer may recur, if free hydrochloric acid persists in sufficient degree, regardless of the surgical procedure employed. In his opinion gastric resection has a place in the treatment of certain of the hemorrhagic duodenal ulcers as well as in the treatment of recurring duodenal ulcer and of gastrojejunal ulceration; it is also applicable in the treatment of patients whose tissue has lowered resistance to the hydrochloric acid in gastric secretion.

In 1924, Charles H. Peck[13] discussed the status of the surgical treatment of chronic duodenal and gastric ulcer at that time, and strongly favored gastro-enterostomy. He contended that it served its purpose as well in those patients without obstruction as those with it. In a total of 196 cases of chronic duodenal ulcer there was a mortality of 8 per cent, and a favorable result, which he considered to mean a lasting clinical cure, in from 80 to 90 per cent. Only one case came to secondary operation for gastrojejunal ulcer, and another for a rigid, painful stoma, while in two other cases there were symptoms suggestive of secondary ulceration, but which were not definitely proved. Peck stressed the word "chronic" in dealing with the subject of the surgical

treatment of ulcer, and he was a firm believer in adequate medical treatment before advising operation.

In 1933, Klein, Aschner and Crohn[14] published a report of a study in the pre- and postoperative gastric secretion in 108 cases in which a subtotal gastrectomy had been performed on the service of Dr. A. A. Berg at Mt. Sinai Hospital (Table I).

TABLE I

DEGREE OF PRE- AND POSTOPERATIVE ACIDITY (*Klein, Aschner and Crohn*[14])

Preoperative Acidity

Number of Cases	Degree of Acidity	Percentage
38 gastric ulcer cases	Normal or subnormal	50
	Hyperacidity	50
210 duodenal ulcer cases	Normal or subnormal	10
	Hyperacidity	90

Postoperative Acidity (early)

Number of Cases	Degree of Acidity	Percentage
35 gastric ulcer cases	Promptly became anacid	77
	Were normal or subnormal	14
	Remained hyperacid	9
197 duodenal ulcer cases	Showed anacidity	38
	Were normal or subnormal	48
	Remained hyperacid	14

In the 108 cases cited above, with adequate follow-up Rehfuss tests over periods of one to eight years, 56 per cent either remained anacid or became anacid while under observation. In one of the gastric ulcer cases which became anacid, recurrence developed, while there were nine jejunal ulcers in the group of duodenal ulcer cases which had not become anacid after gastric resection.

Such a finding materially weakens the argument in favor of subtotal gastrectomy, for, though it is definitely shown that anacidity follows resection in a large percentage of duodenal ulcers, the occurrence of jejunal ulcer in those cases in which free hydrochloric acid persists is higher than has been reported from most clinics following gastro-enterostomy.

Our own experience with primary gastro-enterostomy has been gained from 84 cases. In 12 of these the operation was performed for inoperable carcinoma. They may, therefore, be eliminated from our discussion, leaving 72 cases for consideration. Of these 72 cases, three were not originally operated upon by me, but came under my care for some complication, one with a gastrojejunocolic fistula, and two with symptoms of recurrence. In one of these, twisting of the intestine with adhesions at the site of anastomosis was found, in the other, a ventral hernia, but a normal gastro-enterostomy. Both of these were relieved. There were three cases in the group which had had a previous conservative operation for ulcer: one a closure for perforation, one a pyloroplasty and the third excision of an ulcer. All three had recurrent symptoms with stenosis and were relieved by gastro-enterostomy (Table II).

TABLE II

ANALYSIS OF INDICATIONS FOR WHICH GASTRO-ENTEROSTOMY WAS PERFORMED IN 72 CASES

Indications	Number of Cases
Chiefly ulcer symptoms	39
Chiefly symptoms of obstruction	17
Combined symptoms of ulcer and obstruction	16
Total	72

Well over half the patients were over 40 years of age (Table III).

TABLE III

AGE INCIDENCE

20–30 Years	30–40 Years	40–50 Years	50–60 Years	60–70 Years
18	13	11	23	7

Complications were quite numerous. Those connected with the respiratory system were not serious and none ended fatally. One developed a lung abscess, apparently due to aspiration, and was operated upon subsequently, successfully. In two patients a cardiac infarct was diagnosed. There were two patients with suppurative parotitis, one of whom had to be operated upon.

The complications connected directly with the operation consisted of hemorrhage, vicious circle, and evisceration. In the first patient upon whom I performed a gastro-enterostomy, I gained experience with all three of these complications. Immediately after operation he began to vomit blood, repeatedly and in large quantity. Bleeding was controlled by gastric lavage with hot saline and instillation of dilute adrenalin solution. As the result of vomiting and straining he broke open his abdominal wall, which required secondary suture. Then he developed symptoms of vicious circle which became so severe as to necessitate reoperation. An entero-enterostomy was performed. He has since been entirely free from gastric symptoms. This is the only patient in whom I ever performed a pyloric exclusion at the same time as the gastro-enterostomy. Whether that had anything to do with the complications I do not know, but it made such an impression on me then, that I have never employed the procedure since.

There were two other patients with severe postoperative hemorrhage, one of which ended fatally, apparently due to bleeding from the ulcer. She never vomited blood, but simply became progressively more anemic without any symptoms and expired on the sixth day. There was no autopsy. The third case was a young man, age 29, who within 12 hours after operation vomited large quantities of blood. The bleeding threatened to end fatally, but was controlled by suture ligature of the bleeding points at the anastomosis.

Except in the case cited above, vicious circle did not develop in any patient. There have at times been lesser disturbances suggestive of it, but they have always adjusted themselves.

Evisceration complicated the convalescence in two cases. We are under

the impression that people with a wide epigastric angle and a rigid costal arch have unusual strain put on midline or lateral incisions, and in such cases have frequently employed a transverse incision about two fingers above the umbilicus, from the outer border of one rectus to that of the opposite one. It seems to guard against evisceration.

We have never encountered a definitely diagnosed gastrojejunal or jejunal ulcer in any of the gastro-enterostomies performed by ourselves, but have had two cases in which a suspicion of the presence of one existed. One cleared up entirely and died some 12 years later of carcinoma of the stomach. The other had secondary ulcer symptoms with bleeding and was diagnosed as probable marginal ulcer on roentgenologic examination. He is well at present. We have, however, had considerable experience with one case of secondary ulceration. The patient was admitted with the diagnosis of gastrojejunocolic fistula. His gastro-enterostomy had been performed at another institution ten years previously, at the age of 23, on a presumptive diagnosis of ulcer, based upon a history of stomach upsets since childhood and one attack of vomiting, which contained blood, without any test meal or roentgenologic examination being made. After correction of his gastrojejunocolic fistula the patient was reasonably well for six years, until he developed symptoms of ulcer with obstruction at the pylorus, which later perforated against the anterior abdominal wall and formed a large mass there. This was liberated and the perforation closed transversely. There have been no serious stomach symptoms since, but, during 1937, he was admitted with intestinal obstruction which was found to be due to carcinoma of the ascending colon and required resection.

Three of the 72 cases, or 4.1 per cent, died as the result of the operation. One was due to acute gastric dilatation with cardiac failure, apparently not recognized in time; the second case died of hemorrhage, probably from the ulcer; and the third died of a peculiar dilatation of the cecum, with necrotic ulcers and multiple perforations of its anterior wall. No adequate explanation for this condition has been found. It probably had no relation to the operation or to the lesion for which it was performed.

Most of the patients have been followed since their operation. Some of those of the younger age group, under 30 years, were operated upon many years ago. They have been difficult to follow and several have disappeared from observation. One of them, a patient with what was diagnosed as ulcer of the third portion of the duodenum, recovered completely after gastro-enterostomy, but died within two years of a cancer of the rectum. There has been a suspicion that the duodenal lesion may have been carcinoma. Another of these patients died 12 years after gastro-enterostomy of exophthalmic goiter. An autopsy showed a perfect stoma. All patients were discharged as cured or improved after operation, and remained so for the period of observation. None have returned to us for subsequent treatment but that of course does not mean that all have remained well. Several were of that nervous temperament so often encountered in young patients with ulcer symptoms and it is possible that they have subsequently developed further trouble.

Of the older age groups several have died of intercurrent disease. All others have remained well except the few who developed recurrent symptoms and are mentioned under complications. A few had symptoms of indigestion or nervousness from time to time but not enough evidence of a lesion on examination to warrant the diagnosis of recurrent ulceration. We are under the impression that there has been restitution to health in at least 90 per cent of the patients. The results with gastro-enterostomy performed on ward cases at the Lenox Hill Hospital have been similar. However, the follow-up on these cases is not sufficiently accurate to warrant their inclusion in this report.

Discussion.—A study of the material submitted in this paper in favor of routine partial or subtotal gastrectomy for duodenal ulcer, as well as a consideration of recent literature not quoted here, and the comparison of these with the abstracts of papers in favor of conservative surgery as well as with the reports of our own cases make us feel that there must be some explanation for these strikingly different results and opinions.

Surgeons in favor of partial or subtotal gastrectomy have reached their conclusion on the basis of certain observations which in short are: Failure of gastro-enterostomy to cure in a large percentage of cases; failure to prevent recurrences; and failure to prevent secondary gastrojejunal or jejunal ulcers. Those who do not favor this radical procedure, or rather who see no necessity for adopting it as a routine for the usual uncomplicated case, base their opinion on the generally good results which follow gastro-enterostomy, and on the fact that they do not frequently see recurrences or gastrojejunal or jejunal ulcers.

There must be some explanation for this difference of opinion and particularly difference of recorded observations. All authors may not be thinking and speaking of the same kind of a case. Some may include a large number of acute ulcers in patients of the younger age groups in whom the results may be quite different from those in the more chronic cases with some disturbance of motor function and perhaps lower acidity values. Whether adequate preliminary medical treatment has been a factor or not may play a rôle. Then there is the question of the race of the individual, his general environment, his temperament and his diet which may all have an important bearing on the behaviour of an ulcer. Taking the extremes of reported gastrojejunal and jejunal ulcers, for instance, varying from the low of 3 to 5 per cent, and comparing them with the high of 34 per cent, reported by Lewisohn,[6] makes one wonder. Are the proponents of conservative surgery less keen observers or are they dealing with a different type of case?

In this connection it may be of interest to call attention to a recent article by Schittenhelm[15] which deals with the geomedical variation of types of lesions, and by the part played by racial factors in these differences. It is known that disease processes are vitally influenced by climate, living conditions, food, general environment, *etc.*, in the same way that infections are.

It certainly gives one food for thought when one reads that some clinics

are advocating radical stomach surgery, partly on the basis of the frequency of gastritis in association with ulcer. This is notably true of some German clinics, and the same observation has been made here. The Mayo Clinic investigated this question by sending a commission to Europe and bringing a European investigator over here, in order to study the material at their clinic. It was definitely shown that there is a decided difference in the lesions, and that associated gastritis is infrequently found at the Mayo Clinic, and can, therefore, not be advanced as an argument in favor of radical surgery.

It seems evident that the selection of cases for operation must have a very important bearing on the results. It must make a big difference whether one includes in the study very young people who had a gastro-enterostomy performed for indigestion, hyperacidity, and a suspected early ulcer which sometimes cannot be demonstrated on the operating table, and compares them with the more chronic cases in an older age group, who have a definitely diagnosed and demonstrable ulcer. I have seen operations performed for suspected duodenal ulcer and at the operation no definite lesion could be found. After careful but fruitless search it was decided to do a gastro-enterostomy anyway, in the hope that it would cure the symptoms. There is no doubt that some years ago, and especially at some clinics, gastro-enterostomy was performed without proper indications, sometimes on the presumptive diagnosis of duodenal ulcer. I believe that this is the cause for many of the poor results or late complications which are now advanced as an argument in favor of more radical surgery.

I am afraid that some surgeons are now making the mistake of advocating unnecessarily radical operations for lesions which are amenable to more conservative operative procedures. Their enthusiasm sometimes carries them to a point which in the eyes of more conservative surgeons is not justified. I have seen two resections performed for suspected duodenal ulcer, one here and one abroad, which showed no pathology when the specimen was examined.

My personal feeling is that no patient with symptoms of duodenal ulcer should be subjected to operation unless he has a definitely diagnosed lesion and unless adequate medical treatment has been tried, preferably repeatedly. I do not believe that the diagnosis of duodenal ulcer per se calls for surgery. Very young people especially, with so-called nervous indigestion, hyperacidity, and that entire group of symptoms which so frequently leads to the clinical diagnosis or at least suspicion of ulcer, should not be subjected to operation. Their symptoms are usually of a functional nature and require medical attention. I fully share the views expressed by Peck as well as by Balfour and others, that it is the chronic duodenal ulcer case, the one which has resisted medical treatment and which seems to have no further hope of relief from such treatment, which requires surgery. As shown in the cases reported, many of them have some degree of obstruction associated with the ulcer, either due to spasm, periduodenal inflammation or scar contracture. It is this class of case which furnishes such excellent results after gastro-enterostomy.

To subject such a patient with an uncomplicated duodenal ulcer to a sub-

total gastrectomy seems wrong to me. It appears unsurgical to sacrifice half or more of a normal stomach in order to remove a small ulcer of the duodenum which anatomically and physiologically is separated from it.

Even admitting that jejunal ulcer may follow gastro-enterostomy more frequently than is reported from the large American clinics, should there then not be instituted a medical regimen after gastro-enterostomy to prevent them? It is claimed that it is the acid gastric chyme which, acting on an improperly protected mucous membrane of the duodenum and jejunum, produces ulcer. It is further claimed that removal of the acid bearing area of the stomach will produce anacidity and thereby prevent jejunal ulceration. Laboratory examinations made since these claims were first advanced have shown that, though anacidity is produced in a large number of cases, hyperacidity may persist after a subtotal gastrectomy and jejunal ulcer develop just the same. This would seem to point to the advisability of abandoning partial gastrectomy as a routine procedure for the cure of uncomplicated duodenal ulcer, and to continue to employ gastro-enterostomy.

For complicated cases of duodenal ulcer, especially those with massive hemorrhages, or with excessive and persistent pain which resists medical treatment, or for recurrent symptoms after a conservative operation, radical resection may be indicated, and may be the safest way to restore a patient to health. With the advances which have been made in gastric surgery, and with the modern pre- and postoperative care, resection has become a well standardized procedure, which in the hands of any well trained surgeon yields uniformly good results with a reasonably low mortality.

Technic of Gastro-Enterostomy.—In planning the operation it should always be with the mental reservation that some other procedure may have to be undertaken in order to meet the conditions found. The aim of the operation is to cure the disease and to avoid complications if possible. To keep patients in bed for a few days prior to operation is valuable in order to acclimate them and to be sure that the respiratory, circulatory and excretory systems are in order. The time may be utilized for the administration of fluids and possibly a transfusion, as well as for daily gastric lavage in those patients having obstruction.

With mouth hygiene and modern anesthesia, intrapulmonary complications should be uncommon.

The incision is planned to obtain good access, and to assure a firm, strong abdominal wall. Usually an epigastric incision, just to the left of the median line, answers the purpose, but in patients with a wide epigastric angle and a rigid costal arch, a transverse incision from the outer border of one rectus muscle to that of the other is of value. It permits good exposure, assures a firm abdominal wall without undue strain, and thereby protects against evisceration.

In the operation itself certain technical points are to be observed to guard against hemorrhage, vicious circle and possibly secondary ulceration. One should choose the most dependent part of the stomach, while it lies at rest,

for the anastomosis. By turning the omentum and the transverse colon upward, a place in the transverse mesocolon which lies directly opposite this dependent part of the stomach, and to the left of the colica media, is chosen for the opening. The loop of jejunum close to the duodenojejunal junction is now picked up the way it lies at rest. The most important thing is not to twist it, but to have it lie naturally. This will avoid angulation and possible subsequent stagnation in the loop. If the conception of the cause of jejunal ulcer is correct, namely, that it is due to the acid gastric chyme striking the jejunal mucosa which is unable to protect itself against it, then any stagnation opposite the stoma may favor such a development. Maintaining a free flow of duodenal secretions in the loop will tend to neutralize whatever hyperacid gastric content has been ejected into the jejunum.

The next most important point is to place the sutures properly. The outer row is for approximation of the serosa, and beyond stating that it should be snug there is no special attention required. The inner suture line, however, is important, in that it has to be sufficiently closely approximated to stop bleeding points and to prevent gastric juice from penetrating. For this same reason none of the mucosa should be trimmed, as is occasionally done. It is all needed to protect and cover the cut edges of the intestinal wall. A simple continuous whip-over stitch serves the purpose best. The new stoma is now fastened into the opening in the transverse mesocolon with the aid of interrupted silk sutures which attach the edges to the wall of the stomach, best just above the stoma.

I believe it to be unimportant what suture material is used or whether one uses clamps. Formerly we used silk for inner and outer suture, but during the last several years silk for the outer and chromic catgut No. 0 for the inner suture line. We use the Roosevelt clamp in all suitable cases, but occasionally perform an anastomosis without clamps.

CONCLUSIONS

Gastro-enterostomy is indicated:

(1) In cancer of the pyloric end of the stomach in which radical operation is impossible on account of extensive local involvement metastases, or poor general condition. If successful, it relieves symptoms and prolongs life for a while.

(2) In all benign obstructions of the pylorus due to extensive adhesions, scar contracture from ulcer, stenosis following operations for perforation, or after unsuccessful pyloroplasties.

(3) In pyloric and duodenal ulcer after repeated attempts at medical cure have been unsuccessful.

(4) In gastric ulcers near the pylorus associated with extensive adhesions or stenosis, in which a side-tracking procedure is considered to be of value.

(5) In some gastric ulcers situated so high, or so adherent, that their removal carries with it a serious threat to life.

Gastrectomy is indicated:

(1) In all cancers, if technically possible.
(2) In gastric ulcers:
    (a) As a primary operation because conservative procedures are rarely successful and because of a tendency to cancerous degeneration.
    (b) As a secondary operation where a previous conservative operation has failed or where gastrojejunal or jejunal ulceration has developed.
(3) In duodenal ulcers:
    (a) When repeated hemorrhages, especially massive hemorrhages, have not been controlled.
    (b) After failure of conservative operations.
    (c) For intractable pain.
    (d) Possibly for gastrojejunal or jejunal ulcer.

REFERENCES

[1] Wölfler, A.: Gastroenterostomy. Zentralbl. f. Chir., **8**, 705–708, 1881.

[2] Rydigier, Ludwig Ritter Rydigier von Ruediger: Verh. der Deutsch. Gesellsch. f. Chir., XIII Kongress, 1884.

[3] von Hacker, Victor R.: Magenoperationen on Prof. Billroth's Klinik Toeplitz and Deuticke, Wien, 1886; *Idem:* Langenbeck's Archiv., **32**, No. 3.

[4] Haberer: Anwendungsbreite und Vorteile der Magenresektion Billroth I. Arch. Klin. Chir., **114**, 122, 1920. *Idem:* Zur Frage des Ulcus pepticum jejuni auf Grund aelterer and neuerer Klinischer Erfahrungen. Arch. f. klin. Chir., **119**, 712, 1922.

[5] Berg, A. A.: The Mortality and Late Results of Subtotal Gastrectomy for the Radical Cure of Gastric and Duodenal Ulcer. ANNALS OF SURGERY, **92**, 340, 1930.

[6] Lewisohn, Richard: Gastrojejunal and Jejunal Ulcers. J.A.M.A., **37**, 442; *Idem:* The Frequency of Gastrojejunal Ulcers. Surg., Gynec., and Obst., **40**, 70, 1925.

[7] DeBakey, Michael E.: Peptic Ulceration, Archives of Surgery, **34**, 230–267, February, 1937. *Idem:* The Physiology of Peptic Ulcer. Surgery, **2**: 653–674, November, 1937.

[8] Heuer, George: The Choice of Operations in the Treatment of Peptic Ulcer. N. Y. State Jour. Med., **35**, January, 1935.

[9] Judd, E. Starr, and Hazeltine, Matthew C.: The Results of Operations for Excisions of Ulcer of the Duodenum. Trans. American Surg. Assn., **48**, 151, 1930.

[10] St. John, Fordyce B.: Follow-up Study of the Results in Surgical Therapy for Gastric and Duodenal Ulcer. Trans. Amer. Surg. Assn., 162–166, 1930.

[11] Balfour, Donald C.: Results of Gastroenterostomy for Ulcer of the Duodenum and Stomach. Trans. Amer. Surg. Assn., 146–150, 1930. *Idem:* Peptic Ulcer, Its Surgical Treatment by Conservative Measures. Surgery, **2**, November, 1937.

[12] Walters, Waltman: Should Gastric Resection be Done for Duodenal Ulcer? Surgery, **2**, November, 1937.

[13] Peck, Charles H.: The Present Status of the Surgical Treatment of Chronic Duodenal and Gastric Ulcer. ANNALS OF SURGERY, **80**, 31–41, 1924.

[14] Klein, Eugene, Aschner, Paul W., and Crohn, Burrill B.: The End Results of Partial Gastrectomy for Primary Gastric and Duodenal Ulcers. Studies in Pre- and Post-Operative Gastric Secretion. Trans. Amer. Gastro-Enterological Association, 1933.

[15] Schittenhelm, Prof.: The Importance of Geomedical Knowledge. J.A.M.A., **106**, 1106–1107, 1936.

DISCUSSION OF THE PAPERS OF DOCTORS CUTLER AND EGGERS

DISCUSSION.—DR. CONSTANTINE J. MACGUIRE, JR. (New York) had recently collected the statistics on the surgery of peptic ulcer from the First Surgical Division of Bellevue Hospital. Before reporting these, however, he emphasized that in almost all cases the guide had been the principles emphasized by Doctor Cutler in his presentation; namely, that surgery of peptic ulcer is the surgery of its complications plus impracticability. In confining surgery to such cases one necessarily has a higher mortality, particularly when the patients come to the service physically and economically at the end of their resources, as is the case at Bellevue Hospital.

Since 1920, 173 cases of duodenal ulcer exclusive of acute perforations were operated upon, as reported in the ANNALS OF SURGERY, **107**, 350-358, March, 1938, by Doctor McCreery: One hundred thirty-three of these cases were subjected to simple gastro-enterostomy with or without cauterization of the ulcer or pyloric exclusion. Most of them had cauterization. There were six cases operated upon as a desperate measure during massive, uncontrollable hemorrhage. These were not included in the general statistics; all died, and in Doctor MacGuire's opinion surgery has no place in the acute stage of massive hemorrhage. Seventeen cases died of various causes, giving a mortality in the gastro-enterostomy group a little under 13 per cent. Of the cases followed more than two years—namely, 97—65 were free of ulcer symptoms and 32 showed varying degrees of gastric distress. Twenty-two cases of duodenal ulcer, not including the massive hemorrhage cases, were subjected to partial gastrectomy, with seven deaths, a mortality of approximately 31 per cent. This mortality could have been lowered had there been fewer attempts to resect the duodenum, as it was probably not necessary to remove the ulcer in all cases (Table I).

Pyloroplasty of various types (Horsley, Finney, *etc.*) was performed for duodenal ulcer in 14 cases. Of the 10 followed, five showed good results and five persistence of some symptoms (Table I).

Of 111 cases of gastric ulcer operated upon, 33 were subjected to gastro-enterostomy with two deaths, a mortality of 6 per cent. Of 22 cases followed, 13 showed good and nine poor results (Table I).

Excision of the gastric ulcer alone was accomplished in 10 cases, with one death. Four of the nine cases followed showed good results, and five poor. Gastrectomy was performed for nine ulcers in 66 cases, with 15 deaths, a mortality of 22 per cent. This high mortality was accounted for in the fact that the majority of these patients showed very advanced craters, fixed in the pancreas and sometimes in the liver. Of 45 cases followed, 44 were free of gastric symptoms (Table I).

Nineteen marginal ulcers were operated upon. In 10, the original gastro-enterostomy had been performed on the First Surgical Division of Bellevue, and in the other nine elsewhere. Fifteen of these cases were subjected to gastric resection, a rather formidable feature in these cases which all showed massive adhesions. The ulcers showed a marked tendency to penetrate the posterior wall of the transverse colon. There were three deaths, a mortality of 20 per cent. Of the 12 cases followed, 11 were completely relieved and one had a recurrence. Of the cured cases, however, two were marginal ulcers at the stoma. Of the previous gastric resections, one was performed on Doctor MacGuire's service and one elsewhere. Simple excision of the stoma was undertaken in four cases with one death. Of the three cases followed all have gastric distress (Table I).

Doctor MacGuire's analysis involved 302 individuals subjected to 337

TABLE I

SYNOPSIS OF RELEVANT DATA UPON 302 PATIENTS WITH PEPTIC ULCER, SUBJECTED TO 337 OPERATIVE PROCEDURES

(First Surgical Division, Bellevue Hospital)

DUODENAL ULCERS: 173 cases.

Gastro-Enterostomy	133
(With or without excision, cauterization or pyloric exclusion)	
Deaths in hospital	17
Survivors followed more than two years	97
Free from G. I. symptoms	65
Persistent G. I. symptoms	32
Partial Gastrectomy	22
Deaths in hospital	7
Survivors followed-up	13
Free from G. I. symptoms	12
Persistent G. I. symptoms	1
Pyloroplasty	14
(Usually Horsley)	
Deaths	0
Followed-up. Free from G. I. symptoms	5
Persistent G. I. symptoms	5

GASTRIC ULCERS: 111 cases.

Gastro-Enterostomy	33
Survivors followed-up	22
Deaths	2
Mortality	6%
Free from G. I. symptoms	13
Persistent G. I. symptoms	9
Excision of Ulcer	10
Deaths	1
Survivors followed-up	9
Free from G. I. symptoms	4
Persistent G. I. symptoms	5
Partial Gastrectomy	66
Deaths	15 or 22%
Survivors followed-up	45
Free of G. I. symptoms	44
Persistent G. I. symptoms	1

MARGINAL ULCERS: 19 cases

Original gastro-enterostomy performed on Service in 10 cases out of 166 gastro-enterostomies, or 6 per cent. Original gastro-enterostomy performed elsewhere in nine cases.

Simple Excision of Stoma and Jejunal Resection	4
Deaths	1, or 25%
Survivors followed-up	2
Persistent G. I. symptoms	2
Partial Gastrectomy and Jejunal Resection	15
Deaths	3, or 20%
Followed-up	12
Free from G. I. symptoms	11
Persistent G. I. symptoms	1
Jejunostomies for Duodenal or Pyloric Ulcer	3
Deaths	1, or 33⅓%
Recovered	2
Relief of G. I. symptoms	2
Resection for Massive Hemorrhage	6
Deaths	6

operations. The cause of death was pneumonia in 13, hemorrhage in 8, wound disruption in 7, peritonitis in 5, shock in 5, and the rest died of other causes.

With the exception of the marginal ulcer group, the mortality was high. Another outstanding fact was that of the patients who recovered from the gastric resection only three had recurrences whereas gastro-enterostomy alone gave freedom from symptoms in less than 65 per cent of the cases.

DR. RICHARD LEWISOHN (New York) was in complete accord with Doctor Cutler but not quite so fully with Doctor Eggers. Among the points mentioned by Doctor Eggers was that gastro-enterostomy is a side-tracking operation and that it reduces the acidity in the stomach, which Doctor Lewisohn assumed to mean that high acidity is reduced to a very low minimum. In Doctor Lewisohn's experience gastro-enterostomy does not side-track the food and does not reduce the acidity. Doctor Eggers reported his mortality for gastro-enterostomy somewhere between 4 and 5 per cent. Doctor Lewisohn's mortality for gastric resection in primary gastroduodenal ulcers has always been below 4 per cent, but he admitted selecting his cases just as carefully as Doctor Eggers and Doctor Cutler have done.

Doctor Lewisohn was a little surprised at the lack of careful and detailed follow-up data on Doctor Eggers's patients. Many years ago, at the Mt. Sinai Hospital, gastro-enterostomy was regarded as a satisfactory procedure, until there was instituted a very careful follow-up study including roentgenologic studies.

Doctor Lewisohn felt that every experienced surgeon was performing gastro-enterostomies in about the same way. In proof of this, the facts are that in many parts of the world, including Sweden, Italy and elsewhere, surgeons are obtaining a high percentage of gastrojejunal ulcers after gastro-enterostomy, and he wondered if "conservative" is the right word for the procedure of gastro-enterostomy, and whether it is not really more conservative, in view of the postgastro-enterostomy complications (hemorrhage and recurrent ulcer) to perform a primary, partial gastric resection, which is really the more conservative operation because, in a majority of cases, the cure is definite and permanent.

DR. FREDERIC W. BANCROFT (New York) said that with passing years there had been a great deal of progress with regard to surgery for peptic ulcer, and that the papers presented brought out the real fact or reason for the changing attitude toward this branch of surgery. Fifteen years ago surgery was not being performed upon medical failures in peptic ulcer but for any type of duodenal ulcer that came into a surgical ward. Today the type that comes to surgeons in clinics like that of the Roosevelt or Lenox Hill Hospitals is a medical failure—the patients who have been given adequate medical care over a long period of time without permanent relief. In considering medical treatment it is necessary that a patient should have had an adequate period of rest in bed. With the edema and swelling of the gastrohepatic membrane secondary to lesser curvature ulcer, rest is necessary in order to relieve the tension resultant from the full stomach on an inflamed membrane. It is necessary to place the area of inflammation at about the level of the heart so that venous out-flow is facilitated and edema diminished. If surgery is required, then, for an ulcer that has resisted medical treatment, the surgical procedure is of necessity more radical. The indications for surgery are either repeated hemorrhages, marked indurated ulcerations without retention, which resist medical therapy, or scar tissue

contraction with obstruction. In the first two, probably resection is advisable, while in the last gastro-enterostomy is usually the operation of choice.

Dr. John A. McCreery (New York) thought that the difference of opinion as to the proper procedure in these cases seemed to him to be due largely to the fact that the ideas as to the etiology of peptic ulcer were still vague, and that surgery was a matter of operating upon symptoms which may recur, as the underlying cause was still present. However, the work that is being done seems to be along generally similar lines and gradually, though more or less deviously, the same conclusions were being approached.

Doctor McCreery said that the experience and the trend of thought at Bellevue Hospital followed very closely those outlined by Doctor Cutler. It is believed at Bellevue that no case should be considered surgical in the absence of an acute complication, such as perforation, until prolonged medical treatment has failed. In this connection it is interesting to note that it is the experience of the Gastric Clinic, which is presided over by surgeons, that only about five per cent of their cases ultimately come to operation. These figures cover individuals in the lowest income group, with the least opportunity for proper rest and diet, and should be bettered in the higher income group. It is in the group of cases, with uncomplicated duodenal ulcer, carried along fairly comfortably in the clinic, that gastro-enterostomy, in the past, was more frequently performed than at the present time. It was successful for two reasons: In the first place, it supplied a safety valve in the case of subsequent pylorospasm; and in the second place, the patient who had undergone an operation for ulcer was more apt to be impressed by the seriousness of his disease and the importance of care in diet than the patient who has been carried through repeated attacks on a medical regimen. If one omits this group of cases and considers as operable only those with complications—hemorrhage, obstruction, intractability—it seemed to Doctor McCreery that the percentage of cases in whom relief can be expected from gastro-enterostomy must be distinctly lowered. It was his feeling that in this group, only those with obstruction and low acidity are primarily candidates for gastro-enterostomy. Hemorrhage and intractable pain, especially if due to chronic perforation, seemed to Doctor McCreery to demand a more radical procedure. "Follow-up" in this group of cases is most important and the final results are difficult of estimation. He had recently reported cases of perforation well for ten years who had returned with symptoms of ulcer. In the last month he had seen two cases in whom gastro-enterostomies for duodenal ulcer had been performed in 1924. They had been followed regularly in the clinic for seven years and then closed. Both had developed gastric symptoms during the past few months, 14 years after their original operation. One has a contracted stoma, with retention. The other is still being studied. Cases such as these make it difficult to say when a case is "cured"; and rather force one to agree with Means, when he said last fall, in Chicago: "Moreover if the surgeon is called in and operates, the physician will still have to care for the patient for the rest of his life, because surgery usually does not cure the patient with ulcer any more than does medicine."

Dr. DeWitt Stetten (New York) said that the subject of peptic ulcer was always a live one. To-day agreement is more or less general that peptic ulcer is primarily a medical disease and that every effort should be made to heal the ulcer by medical measures before surgery is resorted to. Agreement is also general that in spite of the most conscientious medical treatment certain cases eventually become surgical. The surgical indications were very

clearly presented by Doctor Cutler. He congratulated Doctor Eggers for having the courage to take up the cudgels in defense of the somewhat discredited operation of gastro-enterostomy, and was inclined to agree, in some respects, with his thesis. Gastrectomy, not necessarily always subtotal, he felt to be definitely indicated for obvious reasons in all surgical cases of chronic gastric or pyloric ulcer when technically feasible. It may also be indicated in an occasional favorable case of early perforation, when the patient's condition warrants the procedure. It is also indicated in chronic infiltrated ulcers of the duodenum, penetrating into the pancreas or toward the liver, and for cases in which there has been profuse hemorrhage from involvement of large blood vessels, provided the technical difficulties of the operation are not too great and the patient's life is not placed in jeopardy by the procedure.

Sometimes a two-stage procedure may be advisable—primary gastro-enterostomy, followed by partial or subtotal gastrectomy, after the patient's condition has improved and some of the inflammatory reaction around the ulcer has subsided. Gastrectomy is usually indicated in secondary marginal ulceration after gastro-enterostomy. For healed ulcers at the pylorus or in the first portion of the duodenum, with cicatricial stenosis, Doctor Stetten believed that posterior, short-loop retrocolic gastro-enterostomy—in preference to pyloroplasty—is the operation of choice, and probably gives the most satisfactory results obtainable in gastric surgery. In this connection he cited a statement that he made in a discussion at a "Symposium on Gastric and Duodenal Ulcer," held before the Clinical Society of Lenox Hill Hospital, in the fall of 1926: "I know of no other operation upon the stomach, not excluding subtotal gastrectomy, in which the immediate postoperative recovery is usually so easy, in which the end-results are so uniformly satisfactory and gratifying, and in which the patient is so permanently and thoroughly cured as by this operation performed for this disease." Doctor Stetten's experience during the past 12 years had not altered his opinion, but rather confirmed it.

Subsequent ulceration in the stomach, pylorus or duodenum, in the jejunum or at the gastro-enterostomy stoma, after gastro-enterostomy for cicatricial stenosis, is in our experience extremely rare, possibly because in these cases the ulcer diathesis has ceased to exist. He also still favored gastro-enterostomy in those cases of duodenal ulcer where resection would be too difficult or too dangerous, especially because of their too close proximity to the papilla of Vater, and in perforated pyloric and duodenal ulcers where a stenosis is produced by the suture closing the perforation, providing the perforation is not older than 12 hours, and the patient's condition is favorable. Although he had not yet made a thorough statistical study of his material, his experience with gastro-enterostomy in properly selected cases of this group, with proper after-cure, had not been unsatisfactory. He admitted having had an occasional recurrence of symptoms, especially of bleeding, and had seen a number of jejunal and marginal ulcerations, but, off-hand, the percentage of jejunal and gastrojejunal ulcers certainly had not exceeded 4.5 per cent, which is the incidence of this complication, established by Lahey and Jordan in a statistical study of nearly 17,000 gastro-enterostomies, performed by different surgeons in various countries, and which is a little more than the statistics of Eusterman and Balfour, namely 3 to 4 per cent. Doctor Stetten said he certainly had seen nothing remotely approaching the 34 per cent frequency that has been reported from the Mt. Sinai Hospital. Incidentally, since subtotal gastrectomy for ulcer has in recent years become so much more popular, and the opportunity has been presented for more extensive follow-up

studies, gastrojejunal and jejunal ulceration also, after this operation, has been found to be much more frequent than was originally supposed to be the case. Sawkoff in a recent compilation from the Russian literature found the incidence to be 4.32 per cent, practically the same as that recorded by Lahey and Jordan, and even more than that noted by Eusterman and Balfour after gastro-enterostomy. It is questionable whether gastro-enterostomy is of any value in unresectable, lesser curvature ulcers.

DR. CARL EGGERS (closing) referred to Doctor Lewisohn's comment, particularly on the lack of detailed follow-up, and explained that this had been omitted simply because it would have made too long a table. The cases are spread out over many years. Those reported in the paper represented Doctor Eggers' personal experience with gastro-enterostomy. He has followed all of his patients, except a few in the early age-group to which he called attention, and who had drifted away, for a very long time. Some of these early cases were of the high strung, nervous temperament, that those who deal with this type of condition are familiar with. They were well when discharged and for a long time after, though a few may not have remained well. The results of the later cases are stated in the paper which Doctor Eggers did not read in full detail.

With regard to side-tracking, Doctor Eggers emphasized that gastro-enterostomy was conceived as a side-tracking operation. It was devised for obstruction of the pylorus and was used at first for carcinoma and later for ulcer. In Doctor Eggers' opinion, gastro-enterostomy to-day gives the very best results in the patients in whom it brings about a side-tracking and shunts out the pylorus because of obstruction. The case with the fistula of the biliary tract which he presented, shows that beautifully; all the food empties through the gastro-enterostomy, one can watch it pass through the stoma.

Doctor Eggers could not concur that primary resection was a conservative operation for duodenal ulcer. Although Doctor Lewisohn has fine results and is to be congratulated, nevertheless when one is teaching this subject one is on dangerous ground to teach that half the stomach—and that is usually not conceded to be enough—should be resected. The mortality will be high in the hands of men who only occasionally see a case of peptic ulcer requiring surgery. Even though a jejunal ulcer does develop in 3 or 4 per cent of the cases, they can be handled by the original surgeon or turned over to another more experienced one. Furthermore, such a patient may at that time be an entirely different type of patient, years after the original gastro-enterostomy, and, therefore, one to be handled in an entirely different way.

Doctor Eggers said most of his patients are of the older age-group; they are not young patients, but people who have had adequate medical treatment, and in these gastro-enterostomy has been a most successful method of treatment. He lost two patients in whom he performed a resection because roentgenographically there was a suspicion of carcinoma and he felt he might have saved them had he performed a gastro-enterostomy only. Gastro-enterostomy usually relieves the patient even if no anacidity is produced. After all, the aim in medicine is not to produce anacidity but to relieve symptoms or to cure the patient. Doctor Eggers stated that he had no objection to resection of the stomach and that he likes the operation very much, but does not believe that routine resection of the stomach is the ideal operation for a duodenal ulcer. Surgeons should cultivate judgment and apply it in each case in order to insure the best results.

POSTOPERATIVE JEJUNAL ULCER

A STUDY OF TWENTY-THREE CASES OPERATED UPON AT THE PRESBYTERIAN HOSPITAL, NEW YORK

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WITH the extensive development of gastric surgery in the past 50 years a new clinical entity has become recognized; namely, postoperative jejunal ulcer. Primary jejunal ulcer, a solitary lesion of the upper jejunum, not preceded by gastrojejunostomy and occurring in the absence of such diseases as typhoid, amebic or bacillary dysentery, cholera, tuberculosis, syphilis and sepsis, is exceedingly rare. Judd[1] has never seen a case and questions its existence. Ebeling,[2] in a thorough review of the subject, concluded that primary jejunal ulcer is a most infrequent lesion. Not so, however, relative to the occurrence of jejunal ulcer following gastric surgery.

The first authentic case of postoperative jejunal ulcer was reported by Braun,[3] in 1889. From that time on, the condition has been diagnosed with increasing frequency, depending upon the interval after operation and the thoroughness with which surgeons have followed their patients subjected to gastro-intestinal surgery. Paterson,[4] in 1909, found but 52 undisputable and ten doubtful cases of jejunal ulcer reported in the literature. Eustermann,[5] in 1920, found 83 cases following various gastric operations performed at the Mayo Clinic. This did not include many cases in which the lesion had healed without requiring surgery. Until recently, it has been felt that jejunal ulcer could be avoided by performing an extensive resection instead of a gastro-enterostomy. Two schools have arisen defending the one operation and condemning the other. The advocates of gastrectomy (von Haberer,[6] Finsterer,[6] Berg,[7] Strauss[8] and others) have estimated the incidence of jejunal ulcer following gastro-enterostomy as varying from eight to 30 per cent. These authors claim a much lower percentage following resection (1 per cent or less). With the increase in popularity of the more radical operation, it has proved to be more productive of jejunal ulcer than was formerly suspected. Balfour,[9] in 1928, reported 28 cases upon which he had operated for jejunal ulcer following gastrectomy. Cames[10] has reported four cases of jejunal ulcer following a procedure resembling the Pólya operation, which is considered a nearly ideal form of gastrectomy. Hurst[11] has collected over 100 cases of secondary ulcer following gastrectomy. Advocates of resection maintain that in such cases the resection may have been inadequate. However, the development of jejunal ulcer has been reported even after subtotal resection (Holst[12]).

It is evident that factors other than the operative procedure employed

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may have some influence on the development of jejunal ulcer. Such factors fall into three groups: (1) Preoperative; (2) operative; and (3) postoperative.

Preoperative Factors.—Of these the outstanding is *the indication for operation.* The more definite the indications for operation in peptic ulcer, the less the likelihood of postoperative jejunal ulcer. Such indications have not yet been infallibly defined (excluding from consideration acutely perforated ulcers). However, the consensus of opinion is that the best results occur in cases that have had a lengthy course of vigorous medical treatment, with symptoms responding less and less to such treatment, and characteristic of the late complications of peptic ulcer, such as obstruction, penetration or uncontrollable bleeding (Moynihan[13]).

Another factor of importance is *the location of the original ulcer.* Since duodenal ulcer as opposed to gastric is more regularly accompanied by hyperacidity (Lindau and Wulff,[14] Hurst and Stewart[15]), it is to be expected that cases of duodenal ulcer coming to operation may be followed by a higher incidence of jejunal ulcer than cases of gastric ulcer. The vulnerability of the jejunal mucosa to highly acid gastric juice has long been recognized (McMaster[16]).

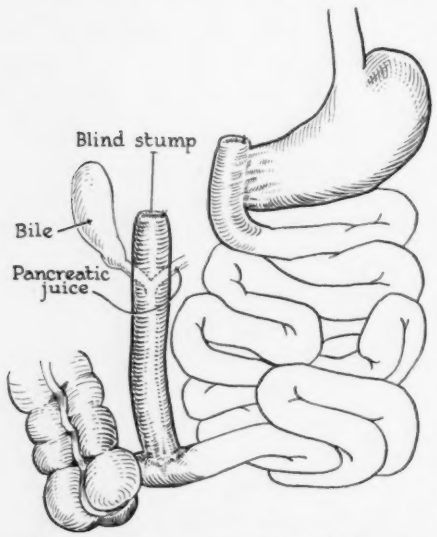

FIG. 1.—Diagrammatic sketch showing how the Mann-Williamson operation deviates the bile, pancreatic juice and duodenal secretion into lower ileum, leaving the jejunum exposed to unneutralized gastric juice.

Operative Factors.—Under this category a number of points must be considered. The most important of these is the degree of neutralization of gastric acidity accomplished by the operative procedure. Moynihan,[13] and Judd[1] maintain that persistent hyperacidity following operation is the most important single cause of postoperative jejunal ulcer. The only reliable way of producing chronic peptic ulcer in animals is to deviate alkaline juices from the site of gastrojejunostomy. This has been done repeatedly by the Exalto[17] procedure, which in this country has become known as the Mann-Williamson[18] operation (Fig. 1).

Such an operation as illustrated in Fig. 1 deviates the bile, pancreatic juice and duodenal secretions from the site of gastrojejunostomy, exposing the jejunum to the action of unneutralized gastric juice. Mann and Williamson[18] found this procedure produced jejunal ulcers in 95 per cent of their dogs. Their results have been confirmed by Ivy and Fauley,[19] Matthews and Dragstedt,[20] Gallagher and Palmer,[21] and many others.

Entero-enterostomy when combined with gastro-enterostomy and the Roux en Y type of anastomosis produces a similar deviation of alkaline juices (Figs. 2 and 3).

Finsterer[17] reported the development of jejunal ulcer in seven out of eight patients upon whom he performed the Roux en Y operation. When the anastomosis was taken down and the original anatomic relations restored, the jejunal ulcer healed in all the cases. Paterson[22] was one of the first to indicate the dangers of Y-anastomosis and entero-enterostomy.

Another factor of importance is the physiologic principle, worked out by Matthews and Dragstedt,[20] that the lower the loop of small intestine the greater its vulnerability on exposure to gastric juice. Accordingly, anterior gastro-enterostomy may be expected to result in a higher incidence of jejunal ulcer than posterior gastro-enterostomy because a lower loop of jejunum comes into contact with gastric juice.

The importance of unabsorbable suture material as a factor in the development of jejunal ulcer is discussed at the end of Case 2.

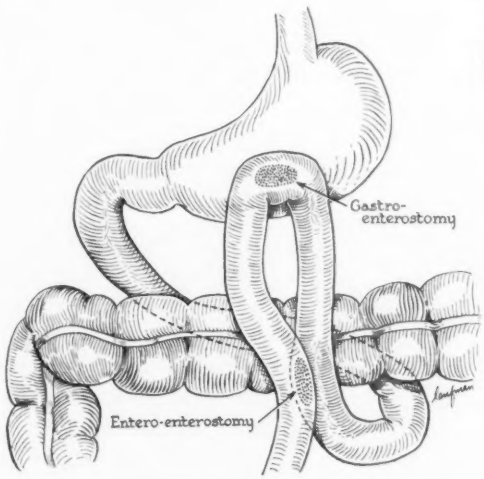

FIG. 2.—Diagrammatic sketch showing how entero-enterostomy when combined with gastro-enterostomy deviates the protective alkaline juices leaving the jejunum at the site of gastrojejunostomy exposed to unneutralized gastric juice.

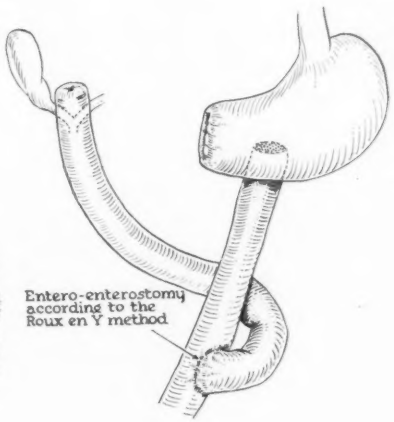

FIG. 3.—Diagrammatic sketch showing how the Y-type of anastomosis produces the same physiologic conditions as the Mann-Williamson operation.

The use of clamps, hematomata of the suture line and traumatic erosions of mucosa at the time of operation cannot be considered significant factors in the development of jejunal ulcer because of the rapidity with which such traumatic lesions heal and the length of time before chronic jejunal ulcers develop (Ivy and Fauley,[19] and Gallagher and Palmer[21]).

Moynihan,[13] and Burden[27] have emphasized the importance of removing the appendix at the time of operation for peptic ulcer. The association of appendiceal disease and pylorospasm and the rôle of pylorospasm in preventing regurgitation of alkaline juices needed for neutralization of gastric acidity has been seriously considered a potential factor in the development of peptic ulcer (Elman[26]). However, the importance of appendicectomy at the time of surgery for peptic ulcer is still a matter of conjecture.

Postoperative Factors.—Following surgery for peptic ulcer numerous fac-

tors come into play which may contribute to the later development of jejunal ulcer. These are mainly matters of diet, habits and latent foci of infection.

Diet.—The natural expectation of a patient just operated upon for peptic ulcer is that the disease is cured and there will be no further need of adhering to a diet. This is far from the true state of affairs. Dietary irregularities are among the most frequent precipitating causes of recurrent symptoms following gastric surgery and may play an important rôle in the later development of jejunal ulcer.

Alcohol.—Alcohol is such an excellent stimulus for the flow of gastric juice that it is occasionally used for estimating gastric function (Ehrmann test meal). For further information concerning the detailed action of alcohol on the gastric mucosa the reader is referred to summaries by Leddig,[28] Himmwich,[29] Kast[30] and Chittenden.[31] Excessive intake of alcohol may influence the formation of postoperative jejunal ulcer.

Tobacco.—Moynihan[13] has demonstrated the increased output and increased acidity of gastric juice after smoking. It is a common statement of ulcer patients that smoking aggravates their symptoms. Circulatory insufficiency in the pyloric area has been considered of some importance in the etiology of peptic ulcer (Reeves,[32] Payr,[33] and Gallagher[34]). The relation of tobacco to vasospastic phenomena (tobacco angina, intermittent claudication in Buerger's disease) has long been recognized. Its relation to jejunal ulcer naturally becomes a matter of interest.

Latent Foci of Infection.—Konjetzny,[35] Turck,[36] Rosenow,[37] Meisser,[38] and others have investigated the possibility that bacteria (particularly the *Streptococcus* and possibly the *Bacillus coli communis*) may be related to the development and persistence of peptic ulcer. Latent foci of infection may be considered as contributory factors in the pathogenesis of postoperative jejunal ulcer.

With the above general principles in mind, 23 cases of postoperative jejunal ulcer operated upon at the Presbyterian Hospital, New York, were studied in minute detail from the time of appearance of the original duodenal or gastric ulcer symptoms, through operation, to the ultimate status of the patients at the present time. In several additional cases the diagnosis of jejunal ulcer had been made at one time or another, but the evidence was not sufficiently conclusive to merit their inclusion in this study. Twenty-three cases is a small number. However, it is hoped that similar studies of postoperative jejunal ulcer may become more frequent, in the hope of gaining a better understanding of this complication of gastric surgery and a method of minimizing its occurrence. Of these 23 cases nine had their primary operations for peptic ulcer at some other hospital.

A number of them illustrate well the physiologic principles involved in the pathogenesis of jejunal ulcer and are, therefore, presented in some detail.

Case 1.—No. 80855: I. H. first experienced severe digestive symptoms at the age of 22. These consisted of gnawing pain in the epigastrium, coming three to four hours after meals, and relieved by food or alkaline powders. Four years after the onset of

these symptoms he was admitted to the Presbyterian Hospital. The gastric contents were somewhat hyperacid (free, 40, total, 80). The Lenhartz diet afforded relief from symptoms. The patient refused to remain on diet; consequently he was operated upon January 21, 1912. The anterior duodenal wall just distal to the pylorus showed a scar. The duodenum was plicated between the pylorus and the site of the ulcer and posterior gastro-enterostomy was performed. He then was well for six years, at which time digestive symptoms recurred. These resembled the preoperative symptoms except that the pain and abdominal tenderness were now situated to the left of the umbilicus. These symptoms responded to diet and the patient remained fairly comfortable until a severe recurrence of abdominal pain 17 years postoperatively. Roentgenograms taken at periodic intervals during these 17 years had been negative. Films taken at this time, however, showed a jejunal ulcer. At operation, August 16, 1929, a gastrojejunocolic fistula was found. The anastomosis was taken down, and the stomach, jejunum and transverse colon were repaired. On the eleventh day the wound disrupted and secondary closure was necessary. The following day intestinal obstruction developed and ileostomy was performed. The patient died the following day.

Comment.—This patient did well for six years after operation. It is possible that the duodenal plication by closing the pylorus created a greater need for the new stoma. It has been claimed that those patients do better after gastro-enterostomy in whom the pylorus is least patent. Not until 17 years postoperatively was there roentgenologic evidence of jejunal ulcer. This illustrates the fallacy of figures based on five- or even ten-year observation following gastric surgery.

Case 2.—No. 85764: W. P. first developed ulcer symptoms at the age of 29. Four years later he was operated upon elsewhere. A duodenal ulcer was found. Posterior gastro-enterostomy and appendicectomy were performed. Within three months his symptoms recurred, and two years later he presented himself at the Presbyterian Hospital because the pain had become unbearable. He was reoperated upon 28 months following the gastro-enterostomy. A linen suture was discovered running through a sinus between gastric and jejunal walls above the stoma with an ulceration at the jejunal orifice of the sinus. The operation consisted in dissolution of the gastro-enterostomy and restoration of the original anatomic and physiologic relationships.

Comment.—In this case, operated elsewhere, jejunal ulcer may have been due to the use of unabsorbable suture material. Moynihan[13] believes that unabsorbed suture material may be a cause of jejunal ulcer. Eustermann[5] found that in one-third of the jejunal ulcer cases on record at the Mayo Clinic unabsorbable suture material had been employed. Woolsey[24] has reported a case similar to this one in which unabsorbed suture material was found hanging from the stoma 21 months after gastro-enterostomy.

Case 3.—No. 242133: T. D., age 41, was admitted to the Presbyterian Hospital because of repeated attacks of vomiting. He was a moderately severe, insulin-treated diabetic, who had been in the hospital several times previously because of acidosis, each attack being accompanied by vomiting. His vomiting at the time of his present admission presented a diagnostic problem. Roentgenologic examination showed 25 per cent six hour retention and suggested obstruction just beyond the pylorus. He was operated upon September 10, 1930; an indurated scar was seen on the anterior surface of the duodenum. The pylorus was patent. A posterior gastro-enterostomy was performed. A roentgenogram taken postoperatively showed the new stoma to be functioning well; no

barium passed through the pylorus. He was well for two years at which time he returned because of uncontrollable vomiting. Fluoroscopy revealed constriction of the efferent jejunal loop and a vicious circle, *i.e.*, reflux of barium into the stomach via the pylorus. Gastric lavage provided only slight relief and he was again operated upon October 14, 1932, two years after his primary gastro-enterostomy. A jejunal ulcer was found 2.5 by 1.5 cm., adherent to the transverse colon and mesocolon. The gastro-enterostomy was taken down; gastric and jejunal openings and mesocolon repaired, and side-to-side jejunojejunostomy performed between the two loops of jejunum on either side of the ulcer-bearing segment. The postoperative course was uneventful. Two months later he returned because of postprandial pain and vomiting. Roentgenologic examination showed reactivation of the old duodenal ulcer and a 20 per cent gastric retention. The symptoms responded to a modified Sippy diet, gastric lavage with removal of the residue at night and belladonna. On this regimen the patient has remained well for over two years after reactivation of the same duodenal ulcer for which he was originally operated upon.

Comment.—This case is unusual because of the rarity with which duodenal ulcer and diabetes coexist.[40] The dietary situation in such cases is extremely difficult to handle. Often, as in this case, when vomiting occurs while the diabetes is not fully controlled, a diagnostic problem arises. In spite of an ideal indication for surgery initially (cicatricial obstruction), jejunal ulcer developed in less than two years. What proportion of the obstruction may have been due to spasm one cannot say, for belladonna was not tried therapeutically. The duodenal ulcer, newly reactivated after dissolution of the gastro-enterostomy, and again with symptoms of obstruction, remains under control with lavage and belladonna. The case suggests that if the indication for operation be obstruction, it is essential to determine by the use of antispasmodics whether cicatricial obstruction or only spasm exists.

Case 4.—No. 47876: M. G. first developed peptic ulcer symptoms at the age of 34. She was treated medically for three months with partial relief. There was no obstruction or evidence of bleeding. Atropine was not tried. She was operated upon October 29, 1920. Duodenal ulcer was visualized. Partial gastrectomy combined with a Y-anastomosis was performed; the appendix was removed. She remained well for three years following operation despite lack of adherence to diet. At this time she returned with a recurrence of her original symptoms. Roentgenologic studies revealed a crater in the jejunal loop, below the stoma, with marked overlying tenderness. After three weeks of Sippy regimen her pain disappeared; fluoroscopy showed the crater persisting but no tenderness. She is still in excellent health, 13 years after her operation. She has been systematically followed by letter and reports occasional, slight digestive discomfort, which has always responded to the administration of an alkali.

Comment.—Despite a Y-anastomosis, this patient is practically free of digestive symptoms, 13 years after operation. The fact that a partial gastrectomy was performed along with the anastomosis may have had some bearing on its success. It is interesting that the one examination of her fasting gastric contents, postoperatively, revealed an anacidity, whereas, usually this type of gastro-enterostomy, without resection, results in hyperacidity.

Case 5.—No. 69908: A. K., age 29, was admitted to the Presbyterian Hospital because of pain of two weeks' duration beginning in the umbilical region and radiating to the right midback. He had been jaundiced five years previously. Gallbladder disease

was suspected. He was operated upon August 7, 1909. The gallbladder was normal. The anterior wall of the stomach, on the lesser curvature, well up near the cardia presented an indurated, puckered scar. Anterior gastro-enterostomy was performed because of a short gastrocolic omentum. The postoperative course was uneventful. The patient remained well for 18 years at which time he was readmitted, seriously ill, presumably due to a perforated jejunal ulcer and was operated upon immediately. A perforated jejunal ulcer was found on the anterior margin of the stoma adherent to the liver. The stoma and ulcer-bearing segment of jejunum were excised and the opening in the stomach repaired. The stomach was then brought through the mesocolon and sutured to the distal loop of the transected jejunum by side- (of stomach) to-end (open end of jejunum) anastomosis. The proximal loop of transected jejunum was anastomosed to the distal jejunal limb about 6 cm. below the gastrojejunostomy. This left essentially a Roux en Y anastomosis without pyloric occlusion. The postoperative course was uneventful. He has adhered to a proper diet and has in general remained well for seven years.

Comment.—Despite anterior gastro-enterostomy, an operation predisposing to subsequent marginal ulcer, this patient went 18 years without digestive symptoms before returning with a perforated jejunal ulcer. This again shows the futility of five- and ten-year follow-up statistics on ulcer cases. Though left with a physiologic arrangement (Roux en Y), which deviates alkaline juices below the stoma, this patient has gone seven years without evidence of a marginal ulcer. The fact that his original ulcer was gastric in location, and quite some distance from the pylorus may bear some relation to his failure to develop jejunal ulcer following a Roux en Y anastomosis.

Case 6.—No. 71401: L. T. first developed peptic ulcer symptoms at the age of 20. He was treated dietetically for five years during which time there were frequent remissions and exacerbations of symptoms. Upon his third admission to the Presbyterian Hospital (over five years after onset of ulcer symptoms) roentgenologic examination showed a 60 per cent residue after six hours. Atropine was not tried. At operation the duodenum was so bound down by adhesions that it could not be seen. The gastrocolic omentum was short; anterior gastro-enterostomy was performed, followed by entero-enterostomy to prevent stasis in the long afferent limb. Within a month he returned because of severe pain beginning to the right of the umbilicus and radiating to the right subcostal margin. Roentgenologic examination revealed a jejunal ulcer 2 cm. below the stoma in the distal jejunal loop. Histamine test showed free acid, 98, total acid, 120. After three weeks of bed-rest and Sippy diet supplemented by moderate doses of atropine, the symptoms disappeared, and control roentgenograms no longer showed evidence of jejunal ulcer. The patient left the hospital only to return for his fifth admission, 16 months later, because of a severe relapse. Roentgenologic examination again revealed the jejunal ulcer previously visualized. It seemed best to remove the ulcer-bearing segment of jejunum in order to prevent acute perforation at a later date. At operation, a jejunal ulcer was found, which had produced adhesions between the ulcerated portion of jejunum and parietal peritoneum. The portion of efferent jejunal limb bearing the ulcer was excised and the cut ends of the gut were inverted. The gastro-enterostomy and entero-enterostomy stomata were patent and were left undisturbed. Convalescence was uneventful. There has been no recurrence of symptoms to date (19 months postoperatively).

Comment.—The outstanding feature of this case is the rapidity with which jejunal ulcer occurred when gastro-enterostomy was combined with entero-enterostomy. It illustrates the greater vulnerability of low loops of

jejunum. Entero-enterostomy undoubtedly contributed to the formation of a jejunal ulcer by deviating alkaline juices from a segment of jejunum needing them most. In this case intensive medical therapy not only relieved the symptoms of the jejunal ulcer but caused the disappearance of the roentgenologic findings in three weeks.

Case 7.—No. 333264: J. W. first experienced symptoms characteristic of peptic ulcer at the age of 16. These continued intermittently for 13 years before he was hospitalized. At the time of his admission to the Presbyterian Hospital in March, 1932, roentgenologic examination showed a deformed duodenal bulb and a 25 per cent gastric retention after six hours. The gastric contents showed free acid, 84, and total, 98, after histamine stimulation. Rest in bed and a Sippy regimen did not relieve the pain. At operation, March 22, 1932, a dense scar was seen on the anterior duodenal wall; posterior gastro-enterostomy with entero-enterostomy was performed. He was discharged, relieved, on the seventeenth day after operation. Ten months later the abdominal pain and nausea returned. Roentgenologic examination at this time showed a jejunal crater halfway between gastro-enterostomy and entero-enterostomy stomata. He was placed on an ambulatory ulcer diet. Three weeks later he was admitted with an acute surgical abdomen and was operated upon immediately. There had been a fresh perforation of a jejunal ulcer into the transverse mesocolon. The perforation was sutured and the abdomen closed. Convalescence was uneventful. To date the patient has remained well.

Comment.—This was an ideal case for surgery; the history was of long standing (13 years), the response to medical treatment was becoming less and less, there was evidence of pyloric obstruction, the gastric contents were hyperacid. However, when entero-enterostomy was combined with gastro-enterostomy jejunal ulcer developed within a year.

Case 8.—No. 397854: A. A. developed symptoms of bleeding peptic ulcer at the age of 24. Roentgenologic studies done elsewhere revealed a duodenal ulcer. He was placed on a Sippy regimen for two months during which time the symptoms were only moderately alleviated. Posterior gastro-enterostomy was then performed elsewhere, in 1927. He remained well for two years at which time his symptoms recurred. He was hospitalized and placed on a Sippy regimen for ten days, and was completely relieved, remaining so for two years. He was again hospitalized elsewhere because of a severe relapse. Roentgenograms indicated pyloric occlusion. He was reoperated upon, and the pyloric occlusion verified. A Billroth II resection was performed. He was then well for two years at which time symptoms recurred, and were worse than any he had experienced before. He was admitted to the Presbyterian Hospital. Roentgenologic examination revealed a marginal ulcer producing moderate obstruction. Medical therapy afforded no relief and the patient was operated upon December 6, 1933. Two centimeters from the distal end of the gastro-enterostomy stoma was an indurated jejunal ulcer which was densely adherent to the transverse mesocolon. The stoma was widely patent and was left *in situ*, the operation consisting of resection of the ulcer, repair of the jejunum and side-to-side entero-enterostomy between the afferent and efferent jejunal limbs. This left essentially a Y-type of anastomosis. The postoperative course was uneventful. Three weeks later the patient returned because of severe postprandial pain. Roentgenograms taken at this time, and repeated a month later, showed a constricted jejunum, a jejunal crater with overlying tenderness, indicative of jejunal ulcer. The pain disappeared after rest in bed, Sippy regimen, atropine and luminal. The patient has not been heard from since.

Comment.—Despite gastrectomy (Billroth II), jejunal ulcer developed within two years. Secondary operation for obstruction from this jejunal ulcer included entero-enterostomy in a jejunum which had already shown itself predisposed to ulcer formation. This deviation of alkaline juices must have played an important rôle in the unusually rapid development of the second jejunal ulcer, three weeks after entero-enterostomy.

Case 9.—No. 59085: B. R. first developed ulcer symptoms at the age of 22. The symptoms could be controlled by dieting and alkali, and it was not until two years later that he was first seen in the Presbyterian Hospital because of an acute attack of epigastric pain and vomiting. Roentgenologic examination revealed a deformed duodenal bulb and 15 per cent retention at the end of six hours. He remained in the hospital for two months and was discharged on an ambulatory ulcer diet. As long as he adhered to it he was well. Unfortunately, his lack of will power led to frequent departures from the diet, each followed by a recurrence of symptoms. He was operated upon April 3, 1925, following a particularly severe episode of pain. A small scar was noted on the lateral wall of the first portion of the duodenum. A posterior gastro-enterostomy with entero-enterostomy was performed. He did not adhere to diet after discharge and six months later returned because of severe postprandial pain. Roentgenologic examination at this time showed a crater in the efferent jejunal limb, 1 cm. below the stoma. During the five years subsequent to the development of this marginal ulcer the patient has been in and out of this hospital and numerous others, leading a miserable life, except during spasmodic periods of adherence to a strict diet, alkali and belladonna.

Comment.—Since gastro-enterostomy entails at least a temporary period of dieting postoperatively, such surgery is hazardous upon an individual who has shown himself unwilling to adhere to medical measures unless extreme indications for operation exist.

Case 10.—No. 250685: M. B. underwent an appendicectomy elsewhere, at the age of 25. The abdominal pain for which he was operated upon persisted, and three months later he was reoperated upon in Dublin for "pyloric obstruction." Posterior gastro-enterostomy was performed. He remained well for two years, during which time he came to this country. He attended the G.I. Clinic of the Presbyterian Hospital from 1926 to 1929, during which time he had many attacks of severe pain in the hypogastrium and left iliac regions, several tarry stools and occasional hematemesis. These symptoms usually responded to dietary measures. Several roentgenologic examinations were made during this three-year interval but failed to show a marginal ulcer until December, 1929, at which time the gastro-enterostomy stoma had closed off completely. He was operated upon January 6, 1930, at which time a scarred jejunum, adherent to transverse colon and mesocolon was found. The operation consisted of taking down the anastomosis, closing the opening in the stomach, resecting the stoma and ulcer-bearing segment of jejunum and reestablishing jejunal continuity by end-to-end anastomosis. One month after discharge from the hospital digestive symptoms recurred. Roentgenologic examination one year after operation showed a reactivation of an old duodenal ulcer. Diet, alkali and belladonna usually afforded relief, but the patient would not adhere long to any regimen. He was, therefore, readmitted for operation June 12, 1934, four and one-half years after operation for the jejunal ulcer. The duodenum was so imbedded in adhesions it could neither be seen nor felt. The transverse mesocolon was short and adherent. It seemed best to section the stomach in the pars media, turn in the distal loop, physiologically excluding the duodenum, and then perform an end-to-side, long-loop, anterior gastro-enterostomy and entero-enterostomy. He convalesced rapidly and left the hospital on the eighteenth day. Two weeks later he returned to the hospital because of low thoracic

pain, thought to be pleuritic in origin. Strapping gave no relief. Roentgenologic examination showed no pleural or pulmonary pathology, but that of the G.I. tract showed a persistent crater just below the gastro-enterostomy stoma in the efferent jejunal limb. With liberal doses of alkali and atropine the symptoms disappeared as did the crater seen roentgenologically. At the present time, the patient has just been readmitted because of a recurrence of severe pain. The jejunal ulcer is again visible roentgenologically.

Comment.—The rapidity with which jejunal ulcer developed in this case illustrates the danger of deviating the gastric contents to a low segment of jejunum, particularly when this segment is deprived of protective alkaline juices by entero-enterostomy. There is an opportunity to compare the length of time it took this patient to develop jejunal ulcer after two different types of gastro-enterostomy. Originally, with a posterior gastro-enterostomy, he went for six years before developing roentgenologic evidence of jejunal ulcer; whereas, after anterior, long-loop gastro-enterostomy combined with an entero-enterostomy, he showed roentgenologic evidence of jejunal ulcer within one month.

SUMMARY.—Of these 23 patients with postoperative jejunal ulcer three were female, 20 were male; this ratio approximates the comparative incidence of duodenal ulcer in the two sexes.

In 21 of the 23 cases the original ulcer was in the duodenum. This corresponds with the impression of other authors that patients with duodenal ulcer are more apt to develop jejunal ulcer than those with gastric ulcer.

The interval between gastro-enterostomy and evidence of existence of jejunal ulcer varied from 12 days to 18 years. In seven cases, more than five years elapsed before the first recurrence of digestive symptoms after operation. In nine cases the first roentgenologic evidence of jejunal ulcer appeared from six to 17 years postoperatively. Such figures suggest the fallacy of end-results of gastric surgery based upon five- or even ten-year follow-up.

The Symptoms and Physical Signs of Jejunal Ulcer.—In two cases acute perforation, two and 18 years after operation, was the first evidence of existence of a jejunal ulcer. Three other cases also experienced acute perforation; in these, however, antecedent symptoms suggestive of jejunal ulcer had been in existence for two, eight and 12 months respectively.

In discussing the symptoms of jejunal ulcer, as exemplified by these 23 cases, it is essential to remember that jejunal ulcer, like duodenal ulcer, probably begins with superficial ulceration of the mucosa, with or without bleeding from the mucosal vessels. This is followed by penetration into the deeper coats, again with or without bleeding, and finally heals with scar tissue formation. Consequently, different groups of symptoms arise depending upon the state of activity in the ulcer.

The symptoms due to bleeding and cicatricial obstruction did not differ markedly in these cases from symptoms characterizing the same states of activity in duodenal ulcer.

It was in the stage of ulcerative and penetrative activity that the jejunal ulcer gave symptoms markedly different from the original ulcer. In general, the site of pain had shifted from the epigastrium to the umbilical region, and in several cases, even lower, to the hypogastrium. The pain radiated to the left lower quadrant, or left flank, or directly through to the back, corresponding to the segmental innervation of the jejunum. The pain was usually much worse than that of the original ulcer, occurred at more frequent intervals, was more continuous and was less relieved by alkali or food. In one case jejunal ulcer was ushered in by severe thoracic symptoms suggestive of pleural irritation. Several of the patients, with no good evidence for the existence of obstruction, complained of severe "gas" which they had not experienced during corresponding stages of activity in their original ulcers.

With the development of gastrojejunocolic fistula (in one case it was asymptomatic) painless diarrhea developed, later followed by the belching of foul-smelling gas. Fecal vomiting was not observed in any of these cases.

On physical examination abdominal tenderness was elicited a good deal more regularly than with the original ulcer. In general the site of maximum tenderness was localized near the umbilicus, either slightly above or below it, and slightly to the right or left of the midline. In those cases with obstruction the enlarged, dilated stomach or duodenojejunal loop could sometimes be palpated.

The Roentgenologic Diagnosis of Jejunal Ulcer.—The roentgenologic signs of jejunal ulcer again depend upon the state of activity of the jejunal ulcer at the time of radiologic examination. The earliest lesions, with only superficial ulceration, may exhibit only tenderness over the stoma or involved segment of jejunum. As the inflammatory process advances, spasm and constriction appear at the site of tenderness. If a considerable number of fluoroscopic examinations are made, the constriction will not appear constantly. This spastic narrowing of the jejunum will frequently disappear if the patient has received sedatives or antispasmodics before examination. With deeper ulceration an actual crater appears. As healing with scar tissue progresses, a real cicatricial obstruction develops which is constant and does not respond to antispasmodics.

In three cases constriction had progressed so far that nothing passed via the stoma; in two instances the obstruction was so marked as to have produced a vicious circle, with barium emptying back into the stomach via the pylorus. Other less frequent signs were marked retraction of the greater curvature above the site of the stoma, exaggeration of the jejunal mucosal folds, overactive peristalsis of the stoma or of the jejunum proximal to the ulcer and marked dilatation of the jejunum above the constriction. In one case, roentgenologic visualization of a gastrojejunocolic fistula was added evidence of the existence of a jejunal ulcer.

It may be remarked, parenthetically, that closure of the gastro-enterostomy stoma does not necessarily mean that the original duodenal ulcer has

healed and that the more natural channel, the pylorus, is being preferentially utilized (Cannon[41]), but may be the result of a cicatricial closure of the stoma following jejunal ulcer.

Gastric Acidity in Jejunal Ulcer.—Of ten cases which had gastric analyses at the time of development of jejunal ulcer (or shortly before roentgenologic evidence appeared) seven had a moderate hyperacidity, either in the fasting contents or in response to a food test meal, two showed a normal acidity and one showed an anacidity (in the one analysis made). It is interesting that this patient had a Roux en Y-resection, followed by jejunal ulcer, which healed under medical therapy and is still asymptomatic, ten years after the operation. It may be that the development of achlorhydria is responsible for her failure to develop another jejunal ulcer.

In five of these cases there was an opportunity to compare the acidity of the gastric contents before operation for duodenal ulcer with the acidity at the time of development of jejunal ulcer. In these cases gastro-enterostomy did not succeed in diminishing the acidity of the gastric juice.

It is unfortunate that only one of the cases in which an entero-enterostomy was combined with a gastro-enterostomy had gastric analyses at the time of the development of the jejunal ulcer. As would be expected, this case showed a marked hyperacidity in the fasting contents as well as after histamine stimulation.

Preoperative Factors Contributory to Jejunal Ulcer Formation: Indications for Surgery

Duration of Symptoms.—Six of the 23 cases were operated upon during their first attack, with antecedent symptoms only one week to three months in duration. In nine of the 23 cases the duration of symptoms was less than eight months.

Duration and Intensity of Medical Treatment.—In only three cases is there evidence that medical treatment was adhered to faithfully for an appreciable length of time. Four cases received practically no medical treatment though in no case was operation considered emergent.

Analysis of Symptoms.—In three cases (originally operated upon elsewhere) the records of the symptoms prior to operation were insufficient to permit any conclusion as to the indication for operation. In seven cases the preoperative symptoms were characteristic of early ulceration, with no evidence of penetration, uncontrollable bleeding or cicatricial obstruction. In at least one case the symptoms were more suggestive of gallbladder disease, and gastro-enterostomy was performed as a matter of routine, upon finding a gastric ulcer at celiotomy.

Of the remaining 12 cases the symptoms suggested penetration alone in two cases, penetration and some degree of obstruction in four cases, and obstruction alone in six cases.

Seven of the ten cases in which some degree of obstruction was suggested

by the symptoms showed definite retention upon roentgenologic examination. Of these ten cases, five had gastric analyses before operation and four showed definite hyperacidity.

In no instance in which obstruction was considered the indication for operation had belladonna (or other antispasmodic) been given a prolonged trial preoperatively. The importance of this is illustrated by Case 3 upon whom gastro-enterostomy was originally performed because of obstruction. The patient subsequently developed a jejunal ulcer and the anastomosis was taken down. Shortly thereafter the original ulcer became reactivated and evidence of obstruction recurred. This time, however, the obstruction responded excellently to large doses of belladonna and gastric lavage. If obstruction be the indication for operation, it is essential to evaluate by lavage, antispasmodics and sedatives how much of the obstruction is cicatricial and how much due to spasm and edema.

Operative Factors Contributory to Development of Jejunal Ulcer

Anterior, Long-Loop Gastro-Enterostomy.—In three cases the original operation was an anterior, long-loop gastro-enterostomy. In two of these entero-enterostomy or Roux en Y drainage was established at the same time. One of these cases had a recurrence of severe digestive symptoms three weeks after operation, and two months later showed definite roentgenologic evidence of jejunal ulcer. The second case went for three years before developing roentgenologic evidence of jejunal ulcer. The ulcer subsequently healed under medical therapy and the patient is still well, ten years after the original operation. The failure of this patient to develop another jejunal ulcer may be due to the fact that partial resection was performed at the time of Roux en Y anastomosis, or possibly to the development of an achlorhydria (her gastric contents having been found anacid on one examination). The third patient, who did not have a simultaneous entero-enterostomy performed, went eighteen years before suddenly perforating a jejunal ulcer. At this time he was operated upon with Roux en Y technic, despite which he has gone for seven years without a recurrence. This may be attributable to the fact that his original ulcer was gastric, supporting the hypothesis that jejunal ulcer is less apt to occur in patients with gastric ulcer.

Three other cases with jejunal ulcers following previous gastro-enterostomy were reoperated and an anterior, long-loop gastro-enterostomy with an entero-enterostomy established. *These three cases developed jejunal ulcer in six months, three and one-half weeks and twelve days respectively.* Entero-enterostomy by preventing adequate neutralization of gastric acidity is dangerous; when combined with anterior gastro-enterostomy, so that a more vulnerable loop of jejunum is exposed to highly acid gastric juice, it is even more so.

Entero-Enterostomy or a Roux en Y Anastomosis.—In nine of the 23 cases entero-enterostomy or Roux en Y anastomosis was the procedure employed. Four of the cases received this type of anastomosis at the time of the operation for the original ulcer. Three of these developed jejunal ulcer in three weeks, ten months and six months respectively. The fourth case had

a partial gastrectomy at the same time and went three years before developing jejunal ulcer.

In five other cases entero-enterostomy was established at a subsequent operation for jejunal ulcer. *Four of these developed their second jejunal ulcer in one month, six months, three and one-half weeks and six months respectively. These same patients had gone for two years, two and one-half years, one year and seven years respectively, before developing jejunal ulcer after their first operation, which in no instance included entero-enterostomy.*

One of these cases came to operation a second time for jejunal ulcer. The entero-enterostomy was left *in situ* but a slightly lower loop of jejunum was anastomosed to the stomach. This time evidence of jejunal ulcer appeared in three months. At the third operation for the jejunal ulcer an anterior gastro-enterostomy using a much lower loop of jejunum, combined with entero-enterostomy, was performed. In twelve days highly acid juice was discharging through a jejunal fistula which had perforated through the abdominal wall. This indicates the danger of anastomosing the stomach to low loops of jejunum and of performing entero-enterostomy or other procedures deviating neutralizing alkaline juices from the gastro-enterostomy stoma.

Gastrectomy as a Protection Against Jejunal Ulcer.—In three cases partial gastrectomy was performed as the original operation for duodenal ulcer. In two of these cases the resection was of the Billroth II type. These two developed jejunal ulcer two and four years after operation respectively. In the third case a Roux en Y anastomosis was established in addition to the resection, and jejunal ulcer developed three years postoperatively.

Four other cases which had developed jejunal ulcer following gastro-enterostomy later underwent gastric resection. In one of these the operation was a Pólya type of resection, and the patient is free of symptoms, six years later. In the second of these four cases a Billroth II gastrectomy was performed and within three and one-half months the patient died of a perforated jejunal ulcer. In the other two cases the Devine type of physiologic exclusion-resection was employed. These two cases should really not be considered as gastrectomies, inasmuch as no actual gastric tissue is removed in this operation. The unusual rapidity with which jejunal ulcer followed exclusion-resection in these cases (three and one-half weeks and 12 days respectively) was undoubtedly attributable to the simultaneous establishment of entero-enterostomy.

It is not intended to discuss here the relative advantages or disadvantages of gastrectomy, but at least in this small group of cases it did not render the patients immune to the later development of jejunal ulcer.

Unabsorbable Suture Material.—In only one case was there reason to believe that unabsorbed suture material might have contributed to the development of jejunal ulcer. In three other cases unabsorbable suture material had been used for the seromuscular stitch, but there was no later opportunity to examine the stoma.

Appendix.—In five cases the appendix was removed at the time of gastro-

enterostomy. In one of these cases, operated elsewhere, the pathologic report was "chronic appendicitis" In 14 of the 23 cases the appendix was not removed, and in those cases in which the gastro-enterostomy was performed at this hospital, it was reported as presenting no evidence of disease at operation. Four patients came to operation for duodenal ulcer despite previous appendicectomy. In comparing the interval of freedom from symptoms following gastro-enterostomy in the groups with and without appendicectomy, no significant differences could be noted. Four of the patients who did not have an appendicectomy still remain well seven to 11 years after their jejunal ulcers have healed, either medically or by secondary operation. One of the cases that did very badly after gastro-enterostomy had undergone an appendicectomy three months before gastro-enterostomy. With so many factors contributing to the pathogenesis of postoperative jejunal ulcer it is naturally impossible to gain precise information as to the exact rôle played by the appendix. From the data in these cases its importance would seem to be minimal and its routine removal at the time of performing gastro-enterostomy of questionable value.

Postoperative Factors Contributory to Development of Jejunal Ulcer

Adherence to Diet.—In 12 cases there was practically no systematized dieting after operation. One patient, operated upon elsewhere, did not receive dietary instructions or precautions upon leaving the hospital. Six of the patients who departed from their diets during the first year after operation experienced a return of digestive symptoms suggestive of jejunal ulcer within the same year. The only two patients who adhered faithfully to a prescribed diet remained well for 14 and 18 years respectively before developing jejunal ulcer. The general impression from a study of the records was that the interval of freedom from digestive symptoms after gastro-enterostomy was proportional to the period of strict adherence to a suitable diet.

Alcohol.—Alcohol played very little rôle in the development of jejunal ulcer in these 23 cases. Most of the patients who had previously enjoyed alcoholic beverages had learned from experience that alcohol and peptic ulcer did not agree. In 17 of the cases there was no evidence of any use of alcohol. In three cases there were definite statements that alcohol was being habitually consumed in at least moderate quantities. Only one patient partook of excessive quantities.

Tobacco.—Twelve of the 23 patients were smoking at least moderately prior to, or at the time of return of digestive symptoms. Some admitted smoking as many as 20, and one patient 30 cigarettes daily. The relationship between smoking and recurrence of digestive symptoms need not necessarily have been a direct one. The excessive use of tobacco may simply have coincided with periods of increased nervous tension, or may have constituted only one phase of a general departure from ulcer regimen.

Latent Foci of Infection.—The following were looked for as possible foci of infection: Teeth; tonsils; sinuses; hemorrhoids; and the genito-urinary tract. In nine cases there were no apparent foci of infection at the time of

the first evidence of jejunal ulcer. The other 14 exhibited one or more potential foci of infection. In no instance, however, were these foci considered important or even contributory factors in the formation of the jejunal ulcer. The evidence concerning the rôle of infection in relation to peptic ulcer is purely speculative and still debatable. However, in view of the mere possibility, that despite our lack of evidence infection may bear even a minimal relation to the development of peptic ulcer, it is worth while eliminating such foci of infection in patients with peptic ulcer.

Healing in Jejunal Ulcer.—It is commonly thought, and rightfully so, that the existence of a jejunal ulcer is a constant threat to the patient's safety. Jejunal ulcers following anterior gastro-enterostomy have been considered more dangerous because of a tendency to perforate freely into the peritoneal cavity; whereas jejunal ulcers following posterior gastro-enterostomy have been considered more likely to become adherent to the transverse mesocolon and colon, with ultimate establishment of a gastrojejunocolic fistula. These impressions were not necessarily borne out by this study. Of five jejunal ulcers operated upon for perforation, two followed anterior gastro-enterostomy. In one of these free perforation had occurred into the peritoneal cavity. Of the other three cases, following posterior gastro-enterostomy, two had perforated freely into the peritoneal cavity. Gastrojejunocolic fistulae ultimately developed in two cases following posterior gastro-enterostomy. The danger of such complications, perforation and gastrojejunocolic fistula might be considered urgent arguments for early operation in jejunal ulcer. However, four patients have now gone from four to ten years after showing evidence of jejunal ulcer, and remain comfortable, except when they indulge in dietary indiscretions. In four cases rigorous dieting produced roentgenologic evidence of complete healing of the jejunal ulcers. In one of these cases the roentgenologic findings reappeared within two years following departure from a suitable diet. The fact that jejunal ulcer like duodenal ulcer may undergo periods of activity and quiescence suggests that if fluoroscopic examinations were made with every return of digestive symptoms following gastro-enterostomy, the incidence of jejunal ulcer might be far greater than is commonly supposed. The fact that jejunal ulcers may heal spontaneously calls for the institution of vigorous dietary measures with alkali, and antispasmodics if necessary, at the slightest recurrence of digestive symptoms.

The Rôle of the Nervous System in Postoperative Jejunal Ulcer.—The importance of mental trauma, emotional unrest, the stress and strain of civilized life have long been known to be among the underlying causes of the development of peptic ulcer. Ivy lamented the fact that dogs do not worry about the stock market and hence are highly refractory to the development of experimental peptic ulcer. Volumes have been written on the importance of psychogenic and environmental factors in this disease, and the reader is referred to an excellent summary on the subject by Cushing.[42]

It was evident that most of these patients either had a good deal to worry about, or were the type to worry a good deal over very little. It is difficult

to compare the environmental factors operating at the time of the formation of the original peptic ulcer with those in existence at the time of development of a jejunal ulcer, and only very general impressions may be obtained. These point to the existence of intricate psychologic and sociologic problems deserving of most thorough study.

Prophylaxis and Therapy of Jejunal Ulcer.—It is obvious that the best treatment of jejunal ulcer is its prevention. This does not necessarily mean performing less gastric surgery. The fact that only 23 cases of postoperative jejunal ulcer could be found in the files of the Presbyterian Hospital is, in itself, a recommendation for gastric surgery. This, of course, with the reservation that absolute indications for surgical intervention must exist. Unwillingness to adhere to medical measures does not constitute an indication for operation. Since gastro-enterostomy entails at least a temporary period of dieting postoperatively, patients who have not adhered faithfully to medical measures are apt to do badly. As for choice of surgical procedure, entero-enterostomy, a Roux en Y anastomosis, or other procedure resulting in the deviation of the alkaline juices from the site of the gastrojejunostomy should be avoided. Since the small intestine becomes increasingly vulnerable to the corrosive effect of gastric juice, the greater the distance from the pylorus, a jejunal loop as close to the ligament of Treitz as is consistent with a good anatomic result should be selected. If the appendix appears diseased it should be removed. The patient should be properly informed of the importance of dietary measures after operation, and should be especially warned to return to the most rigorous type of ulcer regimen with the slightest recurrence of symptoms. Tobacco and alcohol should be forbidden. Foci of infection should be eradicated preferably before operation. And if, despite such precautions jejunal ulcer should still develop, immediate vigorous medical therapy should be resumed, surgery being reserved for the late complications of jejunal ulcer; namely, perforation, gastrojejunocolic fistula, uncontrollable bleeding and cicatricial obstruction. If, on the other hand, the original indications for operation were at all questionable, or if an unphysiologic type of anastomosis was employed, a surgical revision is indicated, with dissolution of the gastro-enterostomy or entero-enterostomy.

The confidence of the patient must be gained by a skillful psychotherapeutic approach and his life so regulated, even to the point of altering his environment, as to eliminate tangible sources of emotional disturbance.

CONCLUSIONS

(1) Twenty-three cases of jejunal ulcer following operations for peptic ulcer have been studied. Nine of these had their primary operation for peptic ulcer elsewhere.

(2) Jejunal ulcer occurred more frequently in males in this series.

(3) Jejunal ulcer occurred more often in patients operated upon for duodenal ulcer.

(4) The interval between gastro-enterostomy and the development of

jejunal ulcer varied considerably, the shortest being 12 days and the longest 18 years.

(5) The symptoms, physical signs, roentgenologic diagnosis and chemistry of the gastric contents in jejunal ulcer are discussed.

(6) The indications for gastric surgery must be definite.

(7) Entero-enterostomy, Roux en Y anastomosis and other procedures leading to inadequate neutralization of gastric acidity should be avoided.

(8) Low loops of jejunum should not be selected (this applies particularly to anterior gastro-enterostomy).

(9) Partial, and even subtotal gastric resection is no guarantee against the future development of jejunal ulcer.

(10) The importance of the postoperative regimen is discussed.

(11) The importance of the psychotherapeutic approach to ulcer patients is discussed.

(12) The prophylaxis and therapy of jejunal ulcer is discussed.

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REFERENCES

[1] Judd, E. S.: Surg. Gynec. and Obst., **33,** 120–126, August, 1921.
[2] Ebeling, W. W.: Annals of Surgery, **97,** 857, June, 1933.
[3] Braun: Der. Verhand. d. Deutsch. Gesellsch. f. Chir., p. 94, 1899.
[4] Paterson H. J.: Annals of Surgery, **50,** 376, 1909.
[5] Eustermann, G. B.: Minn. Med., **3,** 517–524, November, 1920.
[6] von Haberer, quoted by M. E. Blahd: Ohio State Med. Jour., **30,** 156, November, 1934. Finsterer, quoted by M. E. Blahd: Ohio State Med. Jour., **30,** 156, November, 1934.
[7] Berg, A. A.: Annals of Surgery, **92,** 340, 1930.
[8] Strauss, A. A.: J.A.M.A., **95,** 1883, 1930.
[9] Balfour, D. C.: Trans. Am. Surg. Assn., **46,** 232, 1928.
[10] Cames, O.: Rev. de cir. de Buenos Aires, **12,** 891, December, 1933.
[11] Hurst, A. P.: Lancet, **2,** 680, 1928.
[12] Holst, J.: Norsk Mag. f. Laegevidensk, **88,** 111, February, 1927.
[13] Moynihan, B.: Two Lectures on Gastric and Duodenal Ulcer, 1923.
[14] Lindau, A., and Wulff, H.: Surg. Gynec. and Obst., **53,** 621, 1931.
[15] Hurst, A. P., and Stewart, M. J.: Gastric and Duodenal Ulcer. Oxford Medical Publication, London, 1929.
[16] McMaster, P. E.: Arch. Surg., **28,** 825–836, 1934.
[17] Exalto, and Finsterer, quoted by M. E. Steinberg and J. C. Proffitt: Arch. Surg., **25,** 819–848, November, 1932.
[18] Mann, F. C., and Williamson, C. S.: Annals of Surgery, **77,** 409–422, 1923.
[19] Ivy, A. C., and Fauley, G. B.: Am. Jour. Surg., **11,** 531, 1931.
[20] Matthews, W. B., and Dragstedt, L. R.: Surg. Gynec. and Obst., **55,** 265, 1932.
[21] Gallagher, W. J., and Palmer, W. L.: Proc. Soc. Exper. Biol. and Med., 468–473, January, 1933.
[22] Paterson, H. J.: Proc. Roy. Soc. Med. London, **13,** 141–186, 1919–1920.
[23] Renton, J. N.: Glasgow Med. Jour., December, 1923.
[24] Woolsey, G.: Surg. Clin. N. Amer., April, 1934.
[25] Rossler: Mitt. a. d. Grenzgeb. d. Med. u. Chir., **25,** 766, 1913.
[26] Elman, R.: Arch. Surg., **22,** 426, 1931.
[27] Burden, V. G.: Am. Jour. Surg., **21,** 225–239, August, 1933.

[28] Leddig, K.: Deutsch. Arch. f. klin. Med., **150**, 232–250, January, 1926.
[29] Himmwich, H. E.: Alcohol and Man, by Haven Emerson, Macmillan Co., N. Y., 1932.
[30] Kast: Arch. f. Verdauungskr., **12**, 487, 1906.
[31] Chittenden: Am. Jour. Physiol., **1**, 164, 1898.
[32] Reeves, T. B.: Surg. Gynec., and Obst., **30**, 374, 1920.
[33] Payr: Arch. f. klin. Chir., **84**, 799, 1907.
[34] Gallagher, W. J.: Arch. Surg., **15**, 689, 1927.
[35] Konjetzny, G. E.: Zentralbl. f. Chir., **50**, 1026, 1923.
[36] Turck, F. B.: Brit. Med. Jour., **1**, 922, 1907.
[37] Rosenow, E. C.: J.A.M.A., **61**, 1947, 1913.
[38] Meisser, J. G.: Jour. Am. Dent. Assn., **12**, 554, 1925.
[39] Moynihan, B.: Brit. Med. Jour., **2**, 33, 1919.
[40] Crile, G. W.: Diseases Peculiar to Civilized Man, Macmillan Co., N. Y., 1934.
[41] Cannon, W. B.: The Mechanical Factors of Digestion, N. Y., 1911.
[42] Cushing, H.: Surg. Gynec., and Obst., **55**, 1–34, July, 1932.

FUSION IN CHARCOT'S DISEASE OF THE KNEE

A NEW TECHNIC FOR ARTHRODESIS

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A TONE of pessimism pervades the surgical literature dealing with arthrodesis for the treatment of Charcot's arthropathy. Because only an occasional successful fusion has been reported, it seems of value to describe an arthrodesis obtained by means of a technic which has not been previously described.

The method consists of a two-stage operation, the object of the primary procedure being to improve the vascular supply of the involved area through extensive multiple drill-holes. The drill should be at least four inches long and one-fourth of an inch in diameter. The technic consists of placing drill-holes in both femoral and tibial condyles in a fan-shaped distribution so that on each side the drill-point penetrates the cortex in the region of the parosteal bone formation, and in the shaft it should travel for three or four inches into the medullary cavity. The leg is then immobilized by a posterior plaster splint for a period of four to six weeks after which time a standard arthrodesis is performed.

It is well known that there is no actual infection and no toxin present at the site of Charcot's disease. The experimental work of Eloesser showed that neuro-arthropathies can be produced by traumatizing the joints of cats in which the limb has been previously rendered anesthetic by severance of the posterior sensory nerve roots. It would appear, therefore, that the underlying pathologic change is the destruction of the normal protective sensory mechanism of the joint so that it is unable to carry on its normal response to trauma. In a typical involvement of the knee there is primarily diminished sensation, followed by marked effusion, distention of the capsule and relaxation of the ligaments. These changes lead to injury of the articular cartilage and to marginal fractures and sprains with extensive formation of loose bodies. Because of lack of normal reparative processes the bones become sclerotic and there is curtailment of the blood supply. Thinning of the articular cartilage leads to further sclerotic changes in the subchondral articulating areas. Sclerosis may also extend several inches from the joint margin. Concurrently with these joint changes there is a luxuriant growth of irregular masses of parosteal bone which seems to be an effort to repair those injuries which extend through bone to the area of the periosteum. Because of the limited regenerative powers and the avascularity of the bones making up these joints, it seemed

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reasonable to attribute the failure of fusion to a lack of suitable bone tissue. The anticipated change produced by the multiple drilling was verified when five weeks after such drilling of the femur and tibia microscopic studies of sections of bone showed new capillary formation and new fibroblasts and osteoblasts in the channel where the drill had penetrated.

Case Report.—E. M., Italian, male, age 51, was examined, in 1927, by an internist because of paresthesia of the chest. His blood Wassermann was four plus: Intravenous and intramuscular antiluetic treatment was administered between 1927 and 1929. In 1931, while the patient was receiving weekly intramuscular injections of mercury, he began to

FIG. 1.—Preoperative roentgenograms (April, 1937) of a case of Charcot's disease of the knee joint.

develop discomfort, swelling and instability of the left knee. He was examined by the author at that time and a diagnosis of Charcot joint was made. A Thomas walking-caliper brace was applied which allowed him to get about better, but he found it annoying. In spite of continued antiluetic treatment the deformity and swelling of the knee increased until April, 1937, by which time the patient had become totally disabled because of a marked outward bowing of the knee, and the resulting instability so great that he could not walk satisfactorily (Fig. 1). At that time the spinal fluid showed: Wassermann, one plus. Lange, 0012210000. Cell count, 6 per cm. Pressure 180 Mm. Blood Wassermann negative. Urine negative.

Operative Procedures.—April 19, 1937: Under spinal anesthesia, a median parapatellar incision two inches long was made exposing the knee joint. Fifteen drill-holes were placed radially through both condyles of the femur and tibia with a one-quarter inch drill. The leg was then fixed in a plaster splint. On May 28, 1937, an arthrodesis was performed, employing the following technic: A cup-shaped incision six inches in length was made curving just below the patella. Excessive synovia and the fat pad were removed. About one-quarter of an inch of the tibia and of the femur were excised. The tibial spines were refreshened and left protruding through a notch which had been made in the intercondylar fossa. Fixation of the bones was then produced by the introduction of two Steinman pins

which entered through the upper end of the tibia and were introduced into the femur, crossing each other. The leg was placed in a plaster hip spica. The fixation pins were removed five weeks after operation.

Subsequent Course.—Examination on August 25, 1937, showed clinically that the knee was solid. The spica was removed and a posterior plaster splint applied. On November 23,

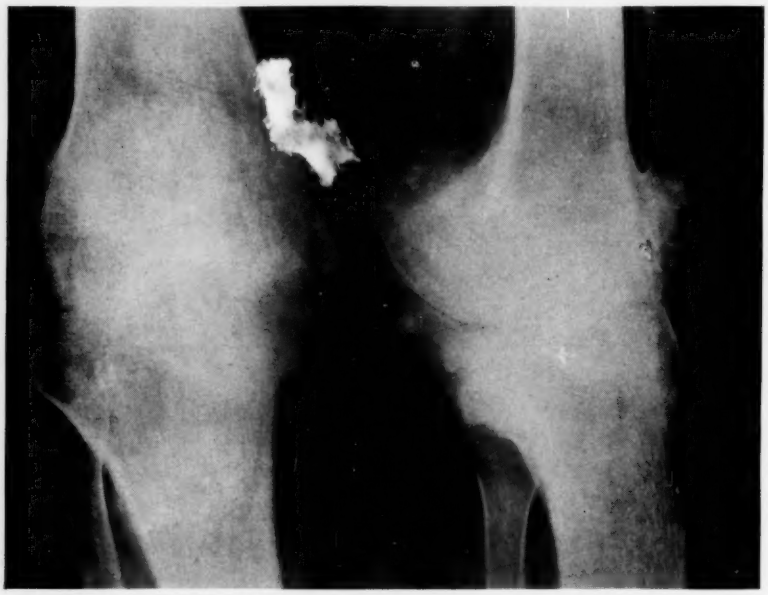

FIG. 2.—Roentgenograms (November 2, 1937) showing the result of the arthrodesis five and one-half months after operation.

1937, union was present, demonstrated both roentgenologically and clinically, and the patient was allowed free weight-bearing.

SUMMARY AND CONCLUSION

A report is made of a rapid and successful fusion in a case of Charcot's disease of the knee by a two-stage operation, the primary procedure consisting of multiple drilling of the involved bones to increase their vascularity and osteogenic power. This procedure was followed five weeks later by an arthrodesing type of operation.

The results of arthrodeses upon neuropathic knee joints have been disappointing. This may be attributed to the poor blood supply and to the sclerosis of the osseous tissue, rather than to any inherent loss in the processes of tissue reaction and regeneration.

INTRAMEDULLARY PRESSURE WITH PARTICULAR REFERENCE TO MASSIVE DIAPHYSEAL BONE NECROSIS

EXPERIMENTAL OBSERVATIONS

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AS A result of Lexer's[1] experimental studies, the anatomic distribution of blood vessels in long bones is now clearly understood. But there has been so little concrete experimental study of the function of these vessels, in the voluminous literature dealing with growth, repair, regeneration, atrophy, necrosis and other disorders of bone, that their fundamental physiologic reactions remain, with few exceptions, unknown.

Axhausen[2] found focal epiphyseal bone necroses associated with resorption and incomplete repair in embolism of epiphyseal vessels. Although Streptococci were cultivated from these lesions he attributed the degenerative change to ischemia rather than bacteria because of the absence of inflammation.

Nussbaum[3] experimentally verified the ischemic nature of the lesions described by Axhausen. Nussbaum's work is of particular interest because it is one of the few dealing with experimental vascular insufficiency of epiphyseal bones.

Müller[4] observed focal necrosis of compact human bone in advanced arterial sclerosis due, he believed, to infarction. He attributed the brittleness of senile bone to these lesions and emphasized the difficulty of producing prolonged bone ischemia by simple arterial occlusion. In no instance did he encounter medullary necrosis.

The experiments of Johnson[5] constitute the first fundamental contribution to our knowledge of the rôle played by each of the three components of the blood vascular system of long bones in bone repair. Johnson concluded that outer cortical bone is supplied and its viability maintained by periosteal vessels; whereas the internal portion of the shaft and metaphyses is supplied and kept viable by the nutrient and metaphyseal vessels.

Brunschwig[6] produced extensive bone and marrow necrosis in experimental animals by actually destroying most of the periosteal, metaphyseal and nutrient vascular connections. In some of his experiments the necrosis involved almost the entire shaft and marrow. Infarction occurred only in animals with unclosed epiphyseal lines, and in no instance was there sequestration.

While Brunschwig had shown that bone and marrow could be killed by extensive interference with their blood supply, Drinker and Drinker[7] first studied blood flow and cytology of living marrow vessels perfused with dif-

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ferent substances at different pressures. They demonstrated the effect of stimulation and interruption of the nerve accompanying the nutrient vessels and the effect of asphyxia on volume flow of blood. Their experiments were conducted, however, under conditions in which collateral circulation of the periosteum and metaphyses was uncontrolled and the state of the epiphyseal cartilages in these experiments is not clearly described.

In 1928, Axhausen[8] and his collaborators advanced the hypothesis that massive necrosis in acute suppurative osteomyelitis is due to septic infarction of bone.

Phemister[9] pointed out that the experimental findings of Johnson and Brunschwig are contrary to this assumption because complete ischemia of a massive segment of diaphyseal cortex would require obstruction of at least two independent vascular networks.

We would suggest, however, that the rigidity of the walls of medullary cavities in long bone, and the close interdependence of volume blood flow and pressure which this anatomic characteristic implies, indicates that massive necrosis of acute suppurative osteomyelitis may be due to ischemia caused by increased intramedullary pressure, which may produce its effect independent of the course and origin of the vessels involved. Studies of the effect of pressure exerted in and upon long bones are infrequently recorded in literature.

Bergman,[10] attempting to ascertain the mechanism by which the pressure of expansile tumors produced its changes, drove expanding pins into the medullary cavities of long bones. Spiral fractures always followed. Employing pegs of diameter slightly less than the medullary cavity, he found extensive, painless lacunar resorption, never involving the whole thickness of the shaft, associated with both periosteal and endosteal proliferation, and concluded that continuously acting pressure from inside the medullary canal produces greater proliferation than absorption. One must credit him with clearly emphasizing the importance of distinguishing changes in growing and adult bone, differentiating between directly and indirectly applied pressure, and indicating the importance of the axis of the bone in which pressure is applied. It is almost paradoxical that this investigator, studying pressure which produces its effect so frequently by disturbances in circulation, should have overlooked the importance of the tremendous damage to medullary circulation which his experimental methods induced.

Robl,[11] studying the effect of externally applied pressure, wrapped the long bones of rabbits with elastic bands and springs, and produced resorption of a thin layer of underlying cortex, greatest at the site of greatest pressure. He did not ascertain to what extent these changes were due to the immediate injury of the periosteal circulation or its subsequent exclusion.

Burkhardt,[12] applying pressure to the whole extremity by wrapping the legs of rabbits and dogs with elastic bands, found that extensive bone necrosis accompanied soft tissue necrosis, that bone is especially susceptible to disturbances in circulation, that the marrow withstands ischemia better than

the compact bone itself, and that bone proliferation occurs only when the damage has been great enough to involve the marrow. While the degree and duration of circulatory exclusion is not quite clear in his paper, it constitutes one of the first purely physiologic studies of the bone blood vascular system. The simplicity of the experimental method is worthy of mention.

It is the purpose of this paper to record experimental results obtained in the study of intramedullary pressure with particular reference to massive diaphyseal bone necrosis.

Part I. Studies of Intramedullary Vascular Pressure

Animals.—Ten dogs, young and old adults, were employed. Some of these were street dogs, other had been fed on regular balanced diets with adequate proteins, salts and vitamins for a period of several weeks before being studied.

Preparation.—The animals in good health were anesthetized with 0.03 Gm. of barbital per kilo intravenously. They were then given 200 mg. Toronto heparin intravenously. The right common carotid was cannulated low in the neck and after shaving and preparing the left thigh, the distal one-eighth of the femoral shaft was exposed without otherwise disturbing the periosteum or its adjacent and contiguous structures.

Procedure.—The periosteum over the anterior surface of the femur was then incised vertically for approximately 2.5 cm., the central portion of the incised periosteum stripped laterally about 0.5 cm. on each side, and the cortex of the femur was then perforated approximately 2 cm. proximal to its distal epiphysis by a 0.75 cm. drill. The marrow was punctured by the perforating drill as it penetrated the cortex. A tapered threaded steel cannula was then turned into the cortical perforation, immediately connected to a mercury manometer; and the pressure, thus obtained, recorded simultaneously with the right carotid arterial pressure on a common base line.

Results.—In eight dogs the marrow cannula pressure obtained in this manner rapidly rose to 30 to 40 Mm. of mercury. Pressure variations in the medullary system occurred simultaneously with carotid arterial pressure variations but were of less magnitude. Even Traube Herring type variations in pressure could be clearly seen in the medullary cannula system. These pressure relationships persisted for the duration of the life of the animal if anticoagulants were repeatedly administered.

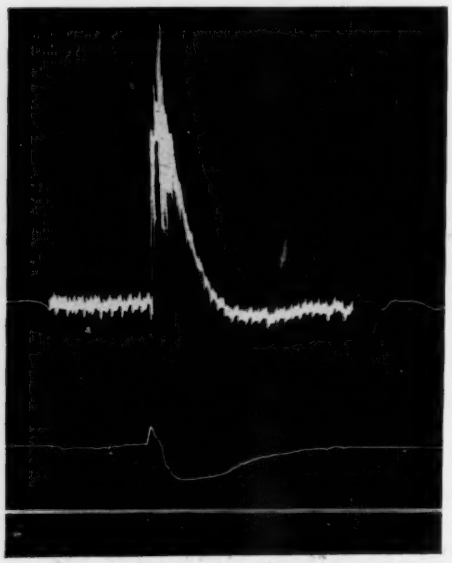
In two dogs the medullary pressure did not rise. In one a clot was found about the bur-hole. In the other the cannula had probably been screwed against the opposite wall.

Conclusions. Part I.—When dogs anesthetized with barbital are heparinized and the marrow cavity cannulated in the manner described, the average relationship of arterial to intramedullary cannula pressure is approximately 3:1 and the intramedullary cannula pressure changes parallel the arterial pressure variations.

Part II. Studies of the Effects of Adrenalin, Ephedrine, Pituitrin and Histamine on Intramedullary Pressure

Adrenalin Hydrochloride.—Experiment. Eight animals, prepared and anesthetized as in Part I, were each given one-half minim of $\frac{1}{1000}$ adrenalin hydrochloride per kilo intravenously.

Results.—In all experiments performed, the following of which is illustrative (Graph 1), essentially the same results were obtained. Synchronous with the rise in arterial tension to 200 plus Mm. mercury, there was a rise of 4 to 6 Mm. of mercury in the intramedullary cannula pressure of only a few seconds' duration, after which the intramedullary cannula pressure precipitously fell from 38 Mm. of mercury to 12 to 14 Mm. mercury where it remained during the period of elevated carotid arterial pressure. Thereafter as the arterial tension fell from 180 to 200 Mm. mercury toward normal, the intramedullary cannula pressure gradually rose so that both the arterial and intramedullary pressures returned to their respective levels which they maintained prior to the administration of adrenalin at the same time. There was no tendency to a delayed over-rise in the intramedullary cannula pressure.



GRAPH 1.—Adrenalin: The upper tracing is the mercury manometer carotid artery pressure. The middle tracing is the mercury manometer medullary cavity pressure. The lowest tracing is the base line. Initial arterial pressure 104, initial intramedullary cannula pressure 34 Mm. mercury. Ten seconds before the point of the abrupt rise in arterial pressure one-half minims per kilo, 1/1000 adrenalin chloride was given intravenously left jugular. Duration of this experiment approximately 20 minutes. Note the abrupt and marked divergence of arterial and intramedullary cannula pressure.

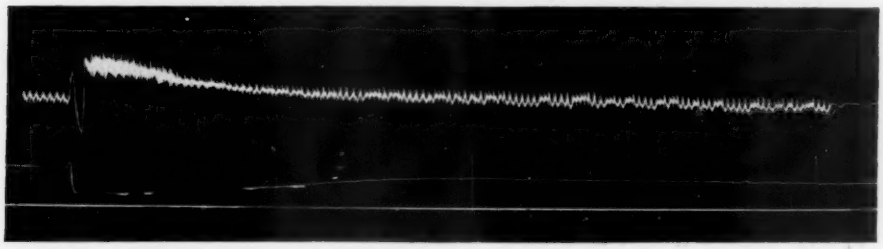
Pituitrin (Surgical).—Experiment. Eight animals, prepared and anesthetized as in Part I, were each given one unit per kilo pituitrin S, intravenously.

Results.—In all of our experiments we obtained essentially the same results as in the following graphically illustrated experiment (Graph 2). Synchronous with the rise in arterial tension from 110–120 to 160 Mm. mercury, there was a very brief rise in intramedullary pressure of 2 to 4 Mm. of mercury followed by an almost instantaneous drop from 36 to 8–10 Mm. mercury. Coincident with the gradual fall in arterial pressure toward normal there was a slow proportionate recovery rise in intramedullary cannula pressure, both arterial and intramedullary cannula pressure arriving simultaneously at the level maintained prior to the administration of pituitrin.

Ephedrine Hydrochloride.—Experiment. Four animals, prepared and

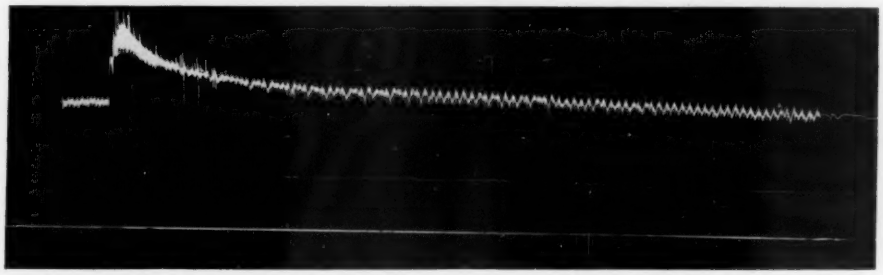
anesthetized as in Part I, were each given 2.5 mg. per kilo ephedrine hydrochloride intravenously.

Results.—The following graphically illustrated experiment (Graph 3) is typical of the results obtained in all dogs studied. Synchronous with the rise in arterial tension from 110–120 to 180 Mm. mercury there was a rise in intramedullary pressure from 34–36 to 52–54 Mm. mercury. This relationship persisted throughout the effective period of the drug so that both the arterial and intramedullary cannula pressure reached the level sustained prior to the administration of ephedrine simultaneously.



GRAPH 2.—Pituitrin: The upper tracing is the mercury manometer carotid artery pressure. The middle tracing is the medullary cavity pressure. The lowest tracing is the base line. Initial arterial pressure 104. Initial intramedullary cannula pressure 36. Ten seconds before the primary rise in arterial pressure one unit per kilo, pituitrin S, was administered intravenously left jugular. The transitory rise in intramedullary cannula pressure followed by the precipitous fall sustained throughout the period of hypertension is apparent. Duration of recorded experiment approximately one hour.

Histamine.—Experiment. Four animals, prepared and anesthetized as in Part I, were each given 0.05 mg. per kilo histamine hydrochloride intravenously.

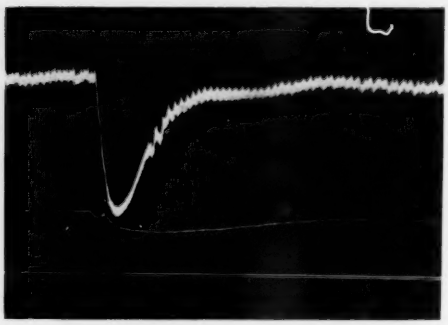


GRAPH 3.—Ephedrine: The upper tracing is the mercury manometer carotid artery pressure. The middle tracing is the mercury manometer intramedullary cannula pressure. The lowest tracing is the base line. Initial arterial pressure 100. Initial intramedullary cannula pressure 38. Ten seconds before the abrupt rise in arterial pressure dog was given 2.5 mg. per kilo ephedrine hydrochloride intravenous left jugular. The precipitous sustained rise in intramedullary cannula pressure throughout the period of hypertension is apparent. Duration of recorded experiment approximately one hour.

Results.—The results in all dogs studied were almost identical with the following graphically illustrated experiment (Graph 4). Coincident with the abrupt fall of arterial pressure from 120 to 30 Mm. mercury there was an abrupt fall in intramedullary pressure from 34 to 24 Mm. mercury, widely disproportionate to the fall in arterial tension. Inconstantly present was a short lived secondary rise in intramedullary pressure after the initial decline.

Comparison of the Pressure Effects of Adrenalin, Ephedrine and Pituitrin S.—Graph 5 is an illustration of the divergent effect of both adrenalin and pituitrin between carotid arterial and intramedullary cannula pressure, contrasted with the parallel effect of both ephedrine and histamine on these pressures.

Conclusions. Part II.—In animals prepared as described in these experiments, the clear-cut, constant divergent effect produced by both adrenalin and pituitrin between arterial and intramedullary cannula pressure and the parallel effect of ephedrine on these pressures are clearly established.



GRAPH 4.—Histamine: The upper tracing is the mercury manometer carotid artery pressure. The middle tracing is the mercury manometer intramedullary cannula pressure. The lowest tracing is the base line. Initial arterial pressure 106. Initial intramedullary cannula pressure 38. Eight seconds before precipitous fall in arterial pressure, dog was given 0.05 mg. kilo histamine hydrochloride intravenous left jugular. The precipitous fall in intramedullary cannula pressure and its gradual recovery are apparent. Duration experiment approximately 25 minutes.

It is not within the scope of this paper to investigate the minute physiologic and pharmacologic reactions which underlie these clear-cut results.

We are well aware that the pressures developed in the intramedullary cannula may not be the exact intramedullary pressure in these experiments. Certainly, however, the fluid in the intramedullary cannula space was in direct pressure continuity with the intramedullary vascular pressure, and the results obtained are in accord with what one would anticipate when dealing with a vascular network enclosed in rigid walls.

Part III. Studies of Degenerative Changes Occurring in Long Bone Following Elevation of Intramedullary Pressure by Physiologic Saline Solution

Experimental Procedure.—Part A: Dogs and pups ranging from 7 to 14 kilos were deeply anesthetized with barbital 25 mg. per kilo, and the anesthesia maintained by repetition of one-half the above dosage of barbital

GRAPH 5.—Shows the results of a composite experiment in which drugs were given in sequence—producing their characteristic change in intramedullary cannula pressure. The precipitous fall and slow rise with adrenalin and pituitrin is in interesting contrast to the sustained rise produced with ephedrine. (a) Adrenalin. (b) Ephedrine. (c) Pituitrin S.

supported by ¼ Gr. morphia whenever the animals reacted sufficiently to whine or move.

TABLE I

GROUP I—BUR-HOLE IN SHAFT OF BONE

Hydrostatic Pressure of 65 to 75 Cm.

Dog No.	Hydrostatic Pressure in Cm.	Length of Time Pressure Was Applied	Number of Cc. Infused	Interval before Dog's Death	Condition of Bone after Death. Other Remarks
4	65	22 hrs.	15 (clot in cannula)	24 hrs.	Dog did not recover from anesthetic. There was no edema or separation of periosteum
2	65	22 hrs.	400 (clot in cannula)	36 hrs.	Dog did not recover from anesthetic. There was no edema or separation of the periosteum
15	65	24 hrs.	400	19 days	On fifth postoperative day wound spontaneously partially disrupted, drained sanguinoseropurulent material 4 days, then gradually healed over. Killed on nineteenth day. There was an irregular thin layer of periosteal new bone deposited over distal half of shaft, the underlying cortical bone was grossly normal and the marrow in the distal half was gray. Epiphyseal cartilages grossly normal
14	65	48 hrs.	2,000	23 days	On sixth postoperative day wound spontaneously disrupted, discharged serosanguineous material for 6 days, then gradually closed. Killed on twenty-third day. There was slight thickening of periosteum around bur-hole. Cortical bone grossly normal. Marrow in immediate vicinity of bur-hole grayish, amorphous, surrounded by plug of granulation tissue. Epiphyseal cartilages normal
101	70	20 hrs.	2,650	7 days	No separation of wound although on third day considerable swelling of soft tissues of entire thigh. Killed on seventh day. Periosteum slightly thickened, easily stripped. No gross defect in cortical bone. Bur-hole marrow defect filled with vascular granulation tissue. Epiphyseal lines closed. Epiphyses normal

Operative Procedure.—The animals were well padded and their restraints loosely applied. The entire thigh and leg were shaved and cleaned and a lateral vertical incision made over the lateral intermuscular septum, the muscles were separated at the septum and two inches of the distal femur

exposed. The lateral periosteum was then incised vertically 3 cm., stripped laterally 0.5 cm. at the midpart of the incision and the underlying cortex was then perforated by a 0.75 cm. drill. The underlying marrow was penetrated as the drill slipped through the cortical bone. A phlanged screw cannula was then turned into the medullary cavity. A pressure apparatus supplying sterile salt solution, 0.9 per cent, was then coupled to the cannula and the wound in muscle, fascia and skin closed with interrupted silk. No attempt was made to close the periosteum.

At the termination of the experiment, the pressure for the first time was lowered or discontinued and the cannula removed. A single skin suture previously placed at the site of the emergence of the cannula was then tied. Collodion dressing was applied and the dog allowed up without splints.

TABLE II

Hydrostatic Pressure of 105 to 135 Cm.

Dog No.	Hydrostatic Pressure in Cm.	Length of Time Pressure Was Applied	Number of Cc. Infused	Interval before Dog's Death	Condition of Bone after Death. Other Remarks
30	105	24 hrs.	350	10 days	No separation of wound although from third to seventh day thigh was extremely swollen. Killed on tenth day. There was diffuse thickening of very vascular, normally adherent periosteum, beneath which there was widespread deposit of new bone intimately adherent to old shaft which when separated from the subperiosteal bone was irregular and motheaten on the opposing surface. There was an area of white bare bone about 2 cm. long on each side of the bur-hole. Marrow irregularly soft, homogeneous, yellow throughout canal. Epiphysis closed, no gross abnormality.
34	135	23 hrs.	6,600	10 days	On fifth postoperative day wound opened spontaneously, drained large amount of purulent material and was granulating. Draining when animal was killed on tenth day. There was diffuse thickening of very vascular periosteum which formed part of the wall of a large extraperiosteal abscess. Diffuse deposit of large amount of subperiosteal new bone intimately continuous with bone of old shaft from which it could be peeled leaving rough mottled surface. Small area bare yellow bone immediate vicinity bur-hole. Marrow irregularly necrotic. Epiphysis closed, no gross abnormality

TABLE III

Hydrostatic Pressure of 180 Cm.

Dog No.	Hydrostatic Pressure in Cm.	Length of Time Pressure Was Applied	Number of Cc. Infused	Interval before Dog's Death	Condition of Bone after Death. Other Remarks
80*	180	12 hrs.	400	14 days	Wound did not disrupt. On fourteenth day when killed there was massive sequestration of distal one-third of shaft, from which the periosteum was separated by a large amount of thin purulent fluid, complete separation from the distal epiphysis and diffuse deposit of new bone over cortex of upper two thirds of shaft, beneath which old cortex was rough but in intimate continuity with newly deposited bone. Marrow totally necrotic in distal one-third, irregularly necrotic mid shaft, and red near upper metaphysis
117*	180	22 hrs.	2,100	16 days	Wound remained closed. Marked swelling and redness until sixth postoperative day. Killed on sixteenth day. Distal one-third of shaft except in immediate vicinity of epiphysis bare and white. Periosteum elevated and separated from dead bone by pus. Epiphyseal line closed. No separation. General deposit new firmly adherent subperiosteal bone remainder of shaft. Marrow necrotic except in spongy bone adjacent to epiphysis
114*	180	20 hrs.	3,400	12 days	Wound healed without disruption. Killed twelfth day. Complete sequestration shaft with separation of periosteum by subperiosteal pus and separation distal epiphysis
47	180	24 hrs.	No appreciable infusion	18 days	Wound infected. Drained very little. Killed on eighteenth day. No bare bone save in immediate vicinity of bur-hole, filled with granulation extending into marrow which was widely necrotic and amorphous. Extensive thinning of old cortical bone by granulation tissue in marrow and deposit of new layer of subperiosteal bone thicker than the original diaphysis. Epiphyseal lines closed and there was marked absorption of end of shaft adjacent, which was replaced by cancellous bone from the epiphysis partly obliterating the distal marrow space

* Mixed flora including *Staphylococcus aureus* obtained from subperiosteal fluid of animals 80, 117 and 114.

TABLE IV

Hydrostatic Pressure of 240 to 265 Cm.

Dog No.	Hydrostatic Pressure in Cm.	Length of Time Pressure Was Applied	Number of Cc. Infused	Interval before Dog's Death	Condition of Bone after Death. Other Remarks
46	256	24 hrs.	? but infusion took place	8 days	Died. Extensive necrosis of bone with separation of both epiphyses and denuded lower one-third of shaft. *Grossly infected.* During 8 of the 24 hours of pressure, tap water was used
37A	240	20 hrs.	1,500	12 days	Marked swelling of thigh from second to sixth day when wound broke open and drained small amount for 3 days. Killed on twelfth day. Entire shaft bare except small area of attachment (?) at linea aspera and at metaphyses, where the periosteum for approximately 1 cm. was adherent to the underlying epiphysis and resorbing shaft through a layer of new bone 0.5 cm. thick. A clear-cut line of separation between the shaft and epiphysis was clearly demonstrable at the cortex but the medulla for approximately 2 cm. was replaced by dense cancellous bone continuous with the epiphyseal bone and new subperiosteal bone deposits. The periosteum itself was greatly thickened and extremely vascular. It contained new bone only where directly approximated to dead shaft near the metaphyses and along the linea aspera
6	265	8 hrs.	?	37 days	Killed on thirty-seventh day. There is tremendous enlargement of the entire shaft. When bisected the thickening is found due to tremendous, diffuse deposit of new, dense subperiosteal bone. The outline of the old shaft is very irregular both on periosteal and endosteal sides. It, in places almost invisible, is still present at the midshaft but near the epiphysis is obliterated by new bone. The marrow cavity is entirely filled with dense spongy bone directly continuous with epiphyseal bone and subperiosteal new bone
73	180	12 hrs.	2,900	11 days	Wound grossly infected from onset. Killed eleventh postoperative day. Large abscess involving deep tissues and communicating with separated periosteum. Distal one-half of shaft bare. Distal epiphysis separated at epiphyseal line

Part B: In this series the same anesthetic and preparation were employed as in animals in Part A. The marrow was cannulated by exposing the femoral condyles and perforating the epiphysis and metaphysis. Pressure was applied as in Part A, and at the termination of the experiment the cannula was removed and the drill hole plugged with wax and fascia. The joint capsule and skin were then closed with interrupted silk.

TABLE V

GROUP 2.—BUR-HOLE IN INTERCONDYLAR REGION

Hydrostatic Pressure of 180 Cm.

Dog No.	Hydrostatic Pressure in Cm.	Length of Time Pressure Was Applied	Number of Cc. Infused	Interval before Dog's Death	Condition of Bone after Death Other Remarks
3	180	24 hrs.	4,000	7 days	Killed. Extensive necrosis of entire shaft. Complete separation periosteum from shaft
6	180	32 hrs.	? but infusion took place	9 days	Died. Extensive necrosis of left femur. Separation of epiphyses and periosteum. Large accumulation of subperiosteal pus
17	180	24 hrs.	? but infusion took place	5 days	Killed. Specimen discarded. No bone necrosis or periosteal change. Bur against opposite wall?
37B	180	24 hrs.	Only small amount	10 days	Killed. Periosteal thickening. No resorption. No separation of epiphysis. Plug of compressed marrow and blood at mid-part of marrow canal. This may have occluded the cannula
2	180	24 hrs.	? but infusion took place	7 days	Killed. Extensive separation periosteum from distal one-half of shaft. No separation epiphysis. Marrow widely destroyed by cannula

Summary. Part III.—When the femoral marrow cavity is infused with physiologic salt solution:

(A) At pressures from 65 to 75 cm. of salt solution for 20 to 24 hours (Table I):

(1) The amount infused ranges from 400 to 2,650 cc.

(2) There is no edema or elevation of periosteum during period of infusion and for 24 hours afterward.

(3) The marrow is killed irregularly distal and adjacent to the site of infusion.

(4) Sequestration of bone does not occur although the wounds are grossly infected.

(5) There occurs a small amount of periosteal new bone especially distal to the site of infusion.

(B) At pressures from 105 to 135 cm. salt solution for 23 to 24 hours (Table II):

(1) The amount infused ranges from 350 to 6,600 cc.

(2) The marrow is killed irregularly throughout most of the cavity.

(3) Sequestration of bone occurs only in the immediate vicinity of the site of infusion even though the wound be grossly infected.

(4) Periosteal new bone is deposited throughout the extent of the shaft, and there is resorption of the underlying cortical bone.

(C) At pressures of 180 cm. or greater salt solution for 12 hours or more (Table III):

(1) The amount of salt solution infused ranges from 400 to 3,000 cc. (Table V).

(2) The marrow becomes completely necrotic.

(3) Massive necrosis of bone with subsequent separation of periosteum and one or both epiphyses occurs. Because of spontaneous reopening of wounds, as subperiosteal fluid accumulated, all wounds studied ultimately became infected in this series. Mixed flora including *Staphylococcus aureus* were recovered from all wounds studied.

(4) Typical involucrum may be formed where sequestration occurs. In two instances (Dogs 6 and 37A, Table IV) sequestration did not occur but a massive segment of old shaft was undergoing substitution when animals were killed.

Conclusions. Part III.—(1) The medullary infusion of 0.9 per cent salt solution at pressures below 75 cm. for 24 to 48 hours does not lead to bone necrosis even though the wounds are grossly infected.

(2) The medullary infusion of 0.9 per cent salt solution at pressures of 180 cm. or greater for as short a period as 12 hours, independent of the amount infused, leads to massive bone necrosis, followed by massive sequestration in bones whose epiphyseal cartilage existed at the time of the experiment, and widespread resorption in bones where epiphyseal lines had closed prior to the experiment. All wounds studied bacteriologically in this series ultimately became infected.

(3) The continued infusion of salt solution, the absence of necrosis or substitution in bones exposed to (55 Mm. mercury) 75 cm. salt solution pressure for 24 hours even when infection follows, and the constant bone substitution or sequestration incurred by application of pressures of (77 Mm. mercury) 105 cm. salt solution or greater leads us to the conclusion that increased intramedullary pressure may be of extreme importance in the production of massive bone necrosis.

(4) In these experiments "stripping" of the periosteum was not a factor in the production of bone necrosis since the periosteum was incised at operation widely enough to allow of decompression throughout the period required for periosteal detachment to occur. Detachment of periosteum is, therefore, secondary to necrosis of the shaft in these experiments and represents the reaction of the living periosteum to the dead infected bone, rather than a primary factor in the production of massive necrosis.

(5) Our experiments suggest that sequestration of dead bone occurs only when there has been complete destruction and long continued exclusion of the vascular connections between bone and surrounding vascular tissue.

DISCUSSION

We are aware that in the experiments cited the marrow cannula fluid was often in continuity with the open marrow vascular system. However, the rapidity with which marrow cannula pressure changes were induced by the drugs administered in Part II as well as the marked variation of fluid infused

FIG. 1.—A roentgenogram taken 18 days postoperative of an experimental animal whose tibia had been subjected to 160 cm. salt solution for 24 hours. (For the sake of clearer roentgenographic reproduction, the tibia above, not included in the foregoing experiments, was used rather than the femur.)

FIG. 2.—A photograph of a typical specimen of massive necrosis produced by high intramedullary pressure (240 cm.) which was removed from Dog 37A, Table IV.

in Part III suggest that the degree of open communication between marrow vessels and cannula fluid in these experiments was relatively insignificant.

In long bones the total medullary capacity in a given bone is fixed by the inexpansile cortex forming its walls except as it is modified by growth or disease. Moreover, excluding the marrow vessels the medullary cavity is occupied by relatively incompressible material. Intramedullary tension must, therefore, be normally dependent upon intravascular tension. Likewise any increase in the relative volume of extravascular tissue can occur only at the expense of the volume of blood in the vascular bed.

In our pressure experiments, therefore, it would seem that whether the circulation in bone and marrow was excluded by compression of vessels or

by substitution of salt solution by infusion is insignificant. And, while part of the exclusion of the circulatory bed in acute suppurative osteomyelitis is probably due to the accumulation of inflammatory exudate in extravascular tissues of bone and marrow, this process is probably of greater importance in zoning pressure heads within the marrow cavity than in the production of absolute ischemia as indicated by the results of Johnson's[6] experiments.

In our opinion the results of the foregoing experiments have an extremely important clinical application in the treatment of acute suppurative osteomyelitis.

Since massive sequestration of diaphyseal bone can occur only secondary to massive bone necrosis, and since massive diaphyseal bone necrosis results from ischemia produced primarily by pressure, the fundamental principle in the treatment of acute suppurative osteomyelitis is *the release of pressure in the infected bone* before the bone is killed.

Failure to clearly distinguish between massive necrosis and sequestration of diaphyseal bone, pointed out years ago by Axhausen,[2] and so important in our present concept of the pathogenesis of sequestration in suppurative osteomyelitis, is responsible for much of the current disagreement as to methods of treatment of acute suppurative osteomyelitis and infected compound fractures.

Once diaphyseal bone necrosis has occurred in the presence of infection, the surrounding living bone and periosteum detach themselves, and depending upon the degree and duration of vascular exclusion, sequestration inevitably ensues. The problem then becomes one not of dealing with acute osteomyelitis, but of sequestration and chronic osteomyelitis. Obviously, since the amount of sequestration parallels the degree and duration of vascular exclusion from the dead bone, preservation of maximum vascular connection with the injured bone and the earliest possible reapposition of vascular tissue to the dead bone is of the greatest importance in treating this phase of the disease.

In acute suppurative osteomyelitis, it is, therefore, necessary that the involved metaphysis be opened early if massive necrosis and subsequent sequestration are to be avoided.

REFERENCES

[1] Lexer: Arch. f. klin. Chir., **71,** 1, 1903; Deutsch. Ztschr. f. Chir., **133,** 170, 1915; Arch. f. klin. Chir., **119,** 520, 1922.

[2] Axhausen: Deutsch. med. Wchnschr., **40,** 1, 111, 1914; Arch. f. klin. Chir., **151,** 72, 1928.

[3] Nussbaum: Bruns. Beiträge z. klin. Chir., **130,** 495, 1923–1924.

[4] Müller: Arch. f. klin. Chir., **142,** 610–612, 1926; Beitr. z. klin. Chir., **138,** 614–624, 1926.

[5] Johnson, J.: Bone and Joint Surg., **9,** 153–184, 1927.

[6] Brunschwig: Proc. Soc. Exper. Biol. and Med., **27,** 1049, 1930.

[7] Drinker, *et al.*: Am. Jour. Physiol., **40,** 514, 1916; Am. Jour. Physiol., **62,** 1, 1922.

[8] Axhausen: Arch. f. klin. Chir., **151,** 72, 1928.

[9] Phemister: Jour. Bone and Joint Surg., **12,** 769, 1930.

[10] Bergman: Arch. f. klin. Chir., **145,** 568–579, 1927.

[11] Robl: Arch. f. klin. Chir., **145,** 515–526, 1927.

[12] Burkhardt: Beitr. z. klin. Chir., **138,** 625, 1927.

THE EFFECT OF THE DIRECT APPLICATION OF COD LIVER OIL UPON THE HEALING OF ULCERS OF THE FEET IN PATIENTS WITH DIABETES MELLITUS

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IN A previous study[1] we found that careful care of the feet of patients with diabetes mellitus decreased the incidence of infection of the feet, improved the condition of the skin and helped in healing the ulcers. In many cases, however, in spite of this treatment, the ulcers failed to heal and constant foot care was necessary to avoid serious infection. It occurred to us that as the tissues of many of these patients were not in an optimum state of nutrition and as vitamin A stimulates the growth of epithelial tissue, that the direct application of cod liver oil might be an effective method of treatment. The healing action of cod liver oil has been reported by several observers who used it in the treatment of wounds, burns, crushing injuries, superficial sores and carbuncles,[2 to 11] with results that the authors felt pointed to an increased rate of healing. Cod liver oil has also been reported to have a bactericidal power.[12, 13]

Observations.—Two groups of diabetic patients have been studied, one a control group of 11 patients who received routine foot care, consisting of daily foot soaks, thorough drying, and the application of lanolin. This routine foot care was carried on for a period of from one to 32 weeks. The second group consisted of 21 patients who had received routine foot care for a period of from one to 136 weeks when cod liver oil was applied locally to the lesions. These patients may be considered as their own controls during the period prior to the use of cod liver oil. Some of the patients in Group II were also in the control group, but the lesions were at different sites, except in Cases 1, 6, and 17.

The routine foot care used in the Clinic has been described.[1] It is administered weekly in the clinic and also daily by the patient at home. When cod liver oil treatment was instituted this foot care was continued, but after drying the feet, gauze saturated with cod liver oil was applied directly to the ulcer and this was kept in place by a noncompressing bandage.

Of the 11 patients in Group I there were four males and seven females. The average age was 59 years. Eight of the patients were on insulin and the average duration of diabetes was 6.7 years. Six had multiple lesions; five required hospitalization.

In Group II all but four of the patients were over 50 years of age. The age in both groups is consistent with previously reported observations[1] in

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TABLE I

EFFECT OF ROUTINE FOOT CARE UPON ULCERS OF EXTREMITIES IN DIABETIC PATIENTS

Group I

Case No.	Age Yrs.	Sex	Description of Lesion	Effect of Routine Foot Care	Onset of Diabetes	Diet in Grams				Remarks
						Carb.	Prot.	Fat	Insulin Daily Units	
1	63	F.	Ulcer and callus right plantar surface	Improved but unhealed after 13 wks.	1932	150	65	85	25	
6	59	F.	Corns right and left great toes. Ulcer fourth right, left toe. Bleb second right toe	Healed after 25 wks. Improved after 8 wks. Healed in 3 wks.	1927	150	65	70	25	Recurred
8	49	F.	Ulcer third left toe (distal)	Improved after 22 wks., not healed	1924	180	65	85	None	Not hospitalized
12	50	M.	Ulcer stump right foot (postoper.)	Improved after 13 wks., did not heal	1927	200	80	100	40	Stump ulcerated again
17	59	M.	2 ulcers right leg, 1 lat. surface right leg	Healed after 14 wks. Unhealed after 28 wks.	1936	180	65	85	None	Hospitalized
19	52	M.	Ulcer third left toe	Healed after 6 wks.	1936	180	70	85	None	Hospitalized
22	65	F.	Hemorrhagic corn fifth right toe	Improved after 17 wks.	1929	150	65	75	20	Recurred
23	65	F.	Corns two, three, five right toes, distal end third left	Healed after 22 and 32 wks., then recurred	1915	180	65	85	35	Hospitalized
24	48	F.	Corn fifth right toe	Unhealed after 24 wks.	1930	150	65	75	55	Hospitalized
25	88	F.	Corn fifth right toe, with cellulitis	Improved after 3 wks.	1930	150	65	75	35	Hospitalized
27	46	M.	Infected corn fifth left, and left great toe and second. Ulcer fourth left toe, and right great toe	Improved Observed Unhealed for Improved 28 Improved wks.	1929	175	75	85	30	All lesions but fifth right toe developed during hospital stay. All improved but left great toe which showed osteomyelitis. Hospitalized

which 81 per cent of the patients with infections or ulcers of the feet were over 50 years of age. Eleven of the patients studied were females and ten were males. The duration of the diabetes varied from one to 17 years. Twelve of the patients required insulin. There were no significant changes in the diet and insulin requirements in the periods before and during cod liver oil therapy, so that these factors did not influence the results. Fifteen of the patients had multiple lesions at the time of this study (Table II). Six of them had to be hospitalized because of the degree of infection present.

Results.—In the control group (Table I) the lesions in Case 1 were improved after 13 weeks but healing did not occur. In Case 6 there was improvement and healing of the lesions, but they recurred later. Cases 8, 12 and 17 improved after 22, 13 and 14 weeks respectively. Case 19 developed ulcers of the same size and type simultaneously on the third left and fourth right toes. One ulcer was treated with routine foot care and the other with cod liver oil. The ulcer on the third left toe, not treated with cod liver oil, healed in six weeks; the ulcer on the fourth right toe, treated with cod liver oil locally, healed in three weeks. Cases 22, 23, 24 and 27 either improved somewhat or remained unhealed after 22 to 32 weeks of treatment. Of the 21 lesions in this group three were unhealed, nine improved, one healed completely and eight recurred.

Of the 21 patients treated with cod liver oil (Table II), only one (Case 16) failed to show complete healing. This patient had generalized arteriosclerosis, coronary sclerosis and evidenced mild congestive heart failure. Continued cod liver oil therapy in the hospital failed to heal the ulcer. Another patient (Case 4) did not heal completely after 44 weeks of cod liver oil therapy in the clinic, but healing resulted in four weeks when bed rest was instituted. In Case 19 there were simultaneous ulcers on the right and left foot, of the same size and type. The ulcer on the left foot was not treated with cod liver oil and healed in six weeks. The ulcer on the right foot treated with cod liver oil healed in three weeks. This patient was in the hospital because of a suspected coronary occlusion, and both ulcers were observed daily.

The average time in weeks required for complete healing of the ulcers, in the 20 cases that healed, was 10.1 weeks. The average duration in weeks of these same ulcers, prior to cod liver oil therapy and during a period of routine foot care, was 24 weeks. These patients during the entire period of observation attended the clinic at least biweekly and during the treatment with cod liver oil, weekly. In four cases the ulcers could really be classified as chronic, having existed for more than 78 weeks.

Before cod liver oil therapy was begun photographs of the feet and the ulcers were made of each patient. Following the institution of cod liver oil follow-up photographs were taken at weekly and later at biweekly intervals. As it is obviously impossible to publish all of these photographs, only some of the cases are shown (Cases 2, 3, 4, 8, 9, 10 and 12).

TABLE II

EFFECT OF TOPICAL APPLICATION OF COD LIVER OIL IN ADDITION TO ROUTINE FOOT CARE UPON ULCERS OF EXTREMITIES IN DIABETIC PATIENTS

Group II

Case No.	Age Yrs.	Sex	Description of Lesion	Duration of Lesion Before C.L.O.,† in Weeks	Vascular State	Healing Time After C.L.O.,† in Weeks	Onset of Diabetes	Diet in Grams			Insulin Daily Units	Remarks
								Carb.	Prot.	Fat		
1	62	F.	Ulcers of right foot, plantar surface and left great toe	24	Good	8	1932	150	65	85	25	Complete healing with C.L.O.
2	65	F.	Ulceration left fourth and fifth toes.	32	Good	14	1935	225	65	85	45	Complete healing with C.L.O.
			Corn third right toe.	12	Good	9						
			Corn third left toe	4	Good	10						
3	65	F.	Bleb tip of right great toe	3	Fair	2	1932	175	65	85	None	Complete healing with C.L.O.
4	55	M.	Ulcer on plantar surface of right great toe	?	Good	48	1936	250	65	85	20	Patient was treated in clinic 44 wks. Ulcer improved but healing was incomplete. Hospitalized and complete healing followed in 4 wks.
5	62	F.	Blebs on second right and left toes	11	Good	4	1929	250	75	85	25	Complete healing with C.L.O.
6	62	F.	Corns right and left two, three, four	11	Good	10	1927	150	65	70	25	Had corns since 1933. Improved with routine care. Started again and markedly improved after C.L.O. Had plantar callus 2 yrs. and 8 mos., which improved somewhat on routine care but healed with C.L.O.
			Plantar callus right and left great toes	11	Good	15						
7	51	M.	Corns right four, five, left fourth	88	Good	12	1933	200	75	85	35	Had corns 88 wks. Improved on routine foot care. Then used salicylic acid causing severe ulcers. Corns healed 12 wks. after C.L.O.
8	49	F.	Corn on plantar surface right great toe.	136	Good	17	1924	180	65	85	None	R.F.C.* helped but C.L.O. resulted in healing

			Ulcer stump third right toe	18	Good	17						
9	71	F.	Ulcers plantar surface both feet and great toes.	118	Poor	L. 24 R. 39	1925	180	65	85	60	Complete healing with C.L.O.
			Ulcer on dorsum of fourth right toe	?	Poor	3						
10	61	F.	Ulcer right great toe.	Unk. 2–3	Good	3	1920	130	60	87	None	First ulcer occurred 4/15/36. Healed in 3 wks. On 9/22/36 lesion recurred which healed in 8 wks. No recurrence in over 1 yr.
			Ulcer right great toe	Unk. 2–3	Good	8					None	
11	45	F.	Gangrene fourth right toe	2	Good	3	1931	200	70	85	30	Complete healing with C.L.O.
12	50	M.	Ulcer third left toe	3	Good	7	1927	200	80	100	40	Complete healing with C.L.O.
13	50	M.	Varicose ulcers both legs	3	Fair	4	1934	180	75	85	None	Complete healing with C.L.O.
14	48	M.	Ulcer left leg anterior surface	24	Good	3	1934	200	75	85	5	2 mos. control on wards before C.L.O.
15	62	F.	Varicose ulcer right leg	25	Good	2	1924	150	75	85	15	Complete healing with C.L.O.
16	67	M.	Ulcer first and second toes left foot—burn	33	Fair	Not healed	1934	180	70	85	None	Ulcers not completely healed after 7 wks. Patient a severe cardiac
17	59	M.	Ulcer lateral surface right leg.	28	Poor	22	1936	180	70	85	None	7 mos. control period with routine foot care. Improvement in 4 wks. with C.L.O., complete healing in 22 wks.
			Ulcer right great toe	1	Poor	14	1936	180	70	85	None	
18	48	M.	Ulcer left great toe. Plantar callus	4	Good	4	1937	180	70	85	45	All lesions healed completely
19	52	M.	Ulcer fourth right toe.	1	Good	2.8	1936	180	70	85	None	Ulcer on left foot, no C.L.O., healed in 6 wks. Ulcer on right foot with C.L.O., healed in 2.8 wks.
			Ulcer third left toe (no C.L.O.)									
20	60	F.	Ulcer right great toe and plantar surface right foot.	28	Good	1	1936	150	70	85	None	Used C.L.O. rarely for 7 mos. 1 wk. in hospital and healed. One readmission. Patient has psoriasis. Ulcer thought to be malignant but healed. No recurrence
			Corn fourth right toe. Ulcer left great toe	28	Good	3.2						
21	51	M.	Ulcer plantar surface of each great toe	78	Good	3	1934	150	70	85	None	Ulcer almost completely healed. Did not return

* R.F.C. refers to routine foot care † C.L.O. refers to cod liver oil

CASE 2.

Before treatment 9/3/36.

1 wk. after treatment 9/10/36.

3 wks. after treatment 9/24/36.

10 wks. after treatment 11/12/36.

14 wks. after treatment 12/10/36.

21 wks. after treatment 1/28/37.

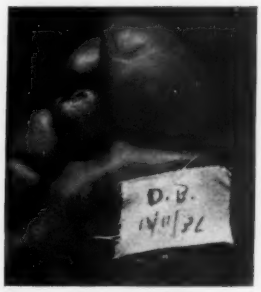
Observing these cases as we did at weekly intervals, it was possible to note the character of the healing that occurred. This healing resulted from a rapid increase in epithelial tissue. As this took place the tissue developed a healthier appearance and the uneven margins smoothed out.

Discussion.—The results in Group I are similar to the results obtained in a larger group treated with routine foot care,[1] in which it was shown that the incidence of infection of the lower extremities and the number of cases requiring hospitalization was decreased.

Case 3.

Before treatment 10/29/36.

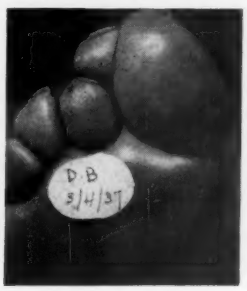
2 wks. after treatment 11/12/36.



7 wks. after treatment 12/10/36.

9 wks. after treatment 12/24/36.

12 wks. after treatment 1/14/37.



19 wks. after treatment 3/4/37.

The results in Group II suggest that cod liver oil will increase the rate of healing of ulcers in patients with diabetes mellitus. What factor in cod liver oil is responsible for this remains open to discussion. It may be that the vitamin content is the responsible agent. On the other hand, cod liver oil contains some highly unsaturated fatty acids[14]—arachidonic, with an iodine number of 334, and clupanidonic with an iodine number of 368. Unsaturated fatty acids apparently have a stimulating effect on the growth of hair and may also have a stimulating effect on epithelial tissue. We are at present studying the effect of cod liver oil and certain of the unsaturated fatty acids on the healing of wounds in rats rendered chronically vitamin A deficient. The results of these studies will, we hope, throw some light on the comparative effect of these two agents.

CASE 4.

Before treatment 8/13/36.

8 wks. after treatment
10/8/36.

13 wks. after treatment
11/12/36.

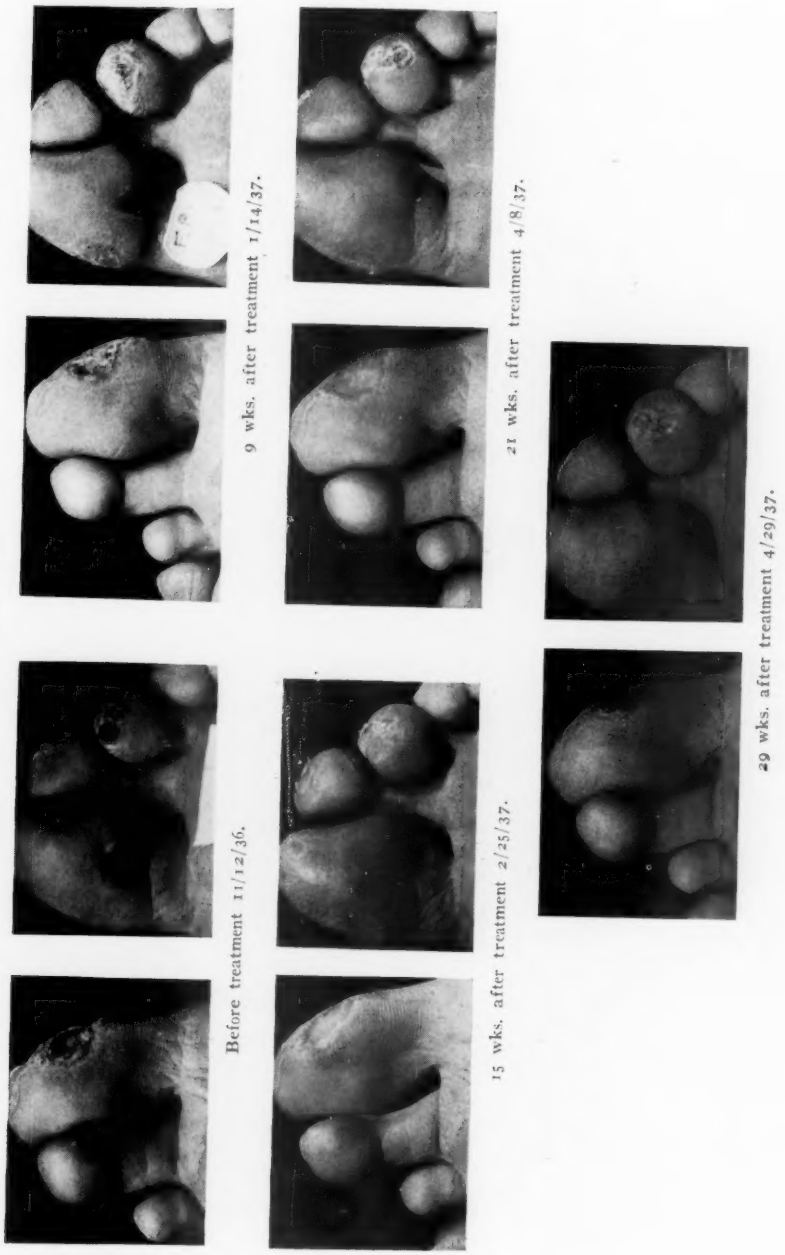
16 wks. after treatment
12/3/36.

26 wks. after treatment
2/11/37.

29 wks. after treatment
3/4/37.

31 wks. after treatment
3/18/37.

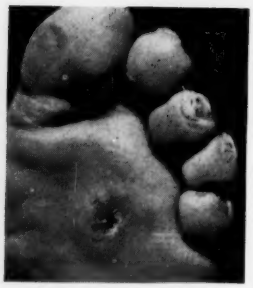
37 wks. after treatment
4/29/37.


CASE 8.
Before treatment 11/12/36.
15 wks. after treatment 2/25/37.
9 wks. after treatment 1/14/37.
21 wks. after treatment 4/8/37.
29 wks. after treatment 4/29/37.

CASE 9.

Before treatment 9/3/36.

3 wks. after treatment
9/24/36.



20 wks. after treatment
1/14/37.

25 wks. after treatment
2/18/37.

29 wks. after treatment
3/18/37.

33 wks. after treatment
4/15/37.

CASE 12.

Before treatment 1/21/37.

2 wks. after treatment
2/4/37.

4 wks. after treatment
2/18/37.

7 wks. after treatment
3/11/37.

11 wks. after treatment
4/8/37.

13 wks. after treatment
4/22/37.

Before treatment 10/1/36.
1 wk. after treatment 10/8/36.
2 wks. after treatment 10/15/36.
3 wks. after treatment 10/22/36.
4 wks. after treatment 10/29/36.
5 wks. after treatment 11/5/36.
6 wks. after treatment 11/12/36.
7 wks. after treatment 11/19/36.

CASE 10.

It seems reasonable to suggest, as a result of these observations, that the application of cod liver oil to ulcers occurring in patients with diabetes mellitus will shorten the healing time and so decrease the incidence of infection of the feet.

SUMMARY

Eleven diabetic patients with ulcers of the feet treated with routine care were studied for periods of one to 32 weeks. In this group, three lesions remained unhealed, nine were improved, one healed completely and eight recurred.

Twenty-one diabetic patients with ulcers of the feet, which had existed for periods varying from one to 136 weeks, were treated by the topical application of cod liver oil in addition to routine foot care.

All of these patients had been treated with routine foot care prior to cod liver oil therapy.

In 20 of these 21 patients complete healing of the ulcers followed the local application of cod liver oil. The average time required for healing to take place was 10.1 weeks.

The average duration of these ulcers prior to this therapy was 24 weeks.

REFERENCES

[1] Brandaleone, H., Standard, S., and Ralli, E. P.: Prophylactic Foot Treatment in Patients with Diabetes Mellitus. ANNALS OF SURGERY, **105**, 120, 1937.

[2] Loehr, W.: Treatment of Extensive Burns of First, Second and Third Degree with Cod Liver Oil. Chirurg., Berlin, **6**, 233, 1934.

[3] Loehr, W.: Cod Liver Oil for External Use in Therapy of Wounds. Deutsch, med. Wchnschr., **60**, 561, 1934.

[4] Loehr, W.: External Application of Cod Liver Oil. Therap. d. Gegenw., **75**, 444, 1934.

[5] Loehr, W.: Combination of Cod Liver Oil and Plaster Bandage in Therapy of Injuries with Tissue Losses in Fingers and Toes. Chirurg., **6**, 5, 1934.

[6] Loehr, W., and Treusch, K.: Treatment of Poorly Healing Ulcerating Stumps with Large Tissue Defects by Cod Liver Oil and Plaster-of-Paris Casts. Zentralbl. f. Chir., **61**, 1686, 1934.

[7] Steel, J. P.: Cod Liver Oil Treatment of Wounds. Lancet, **2**, 287, 1935.

[8] Lauber, H. J.: Relation Between Vitamins and Healing of Wounds. Beitr. z. klin. Chir., **161**, 565, 1935.

[9] Lauber, H. J.: Importance of Vitamins in Healing of Surgical Wounds. Deutsch, med. Wchnschr., **60**, 569, 1934.

[10] Tennent, J. H.: Cod Liver Oil Treatment of Wounds. East African Med. Jour., **12**, 325, 1936.

[11] Daver, M. B.: Crude Cod Liver Oil in Treatment of Wounds. Brit. Med. Jour., **1**, 169, 1937.

[12] Loehr, W., and Treusch, K.: Bactericidal Effect of Cod Liver Oil and Cod Liver Oil Salve on Pyogenic Bacteria—Experimental Studies. Zentralbl. f. Chir., **61**, 1807, 1934.

[13] Tumanskiy, V. K., and Yatsevich, I. A.: Cod Liver Oil Treatment of Infected Wounds. Klinicheskaya Meditsina, Moscow, **13**, 1421, 1935.

[14] Winton, A. L., and Winton, K. B.: The Structure and Composition of Foods. Vol. 3, John Wiley and Sons, 1937.

BRIEF COMMUNICATIONS AND CASE REPORTS

ACUTE PERFORATION OF A DUODENAL DIVERTICULUM

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Case Report.—A white male, age 54, was admitted to the University of Kansas Hospitals, service of Dr. Thomas G. Orr, April 21, 1937, stating that while at work 22 hours previously he had suffered a sudden severe cramp-like pain in the midabdomen just above the umbilicus. Soon afterward he became nauseated and vomited bile-stained fluid several times. He was taken home in an ambulance and hot and cold packs applied to the abdomen with no amelioration of symptoms. His bowels had not moved during the day and several enemata were given with no return of feces or relief of the pain. About three hours after the onset the low epigastric pain became generalized and remained so thereafter.

The patient denied having had any similar seizures in the past and, in fact, denied ever having had any previous gastro-intestinal symptoms whatever.

Physical Examination revealed a rather poorly nourished middle-aged, white male, apparently acutely ill, lying in bed with thighs flexed complaining of severe generalized abdominal pain. Temperature, 100° F.; pulse, 92; respirations, 22; blood pressure, 130/70. The head, neck, and thorax were essentially normal. The abdomen was slightly distended and moved very little with respiration. The entire abdominal wall was tense with board-like rigidity on the right, more marked in the right lower quadrant, where the tenderness was greatest. Hemoglobin, 92 per cent; W.B.C., 10,000; 84 per cent polymorphonuclears. The blood chemistry was normal.

The patient presented the picture of a ruptured abdominal viscus. An acute appendicitis with perforation was considered to be the most probable diagnosis, although perforation of a peptic ulcer and diverticulitis of the colon with rupture were considered.

Operation.—Fibrous adhesions were present between the viscera and the anterior abdominal wall from the region of the pylorus downward into the pelvis. The appendix was thickened and bound down by fibrous adhesions, but no evidences of acute infection were seen. There was free pus in all parts of the peritoneal cavity. Purulent exudate was most marked about the pylorus. The lesser peritoneal cavity was opened and found to contain pus and necrotic tissue. No gastric or intestinal perforation could be identified. The findings, however, suggested a perforation into the lesser peritoneal cavity. The abdomen was closed and drains placed to the lesser peritoneum and right iliac fossa. Cultures from the peritoneal exudate showed B. *coli* and a small gram-positive Diplococcus. The patient responded poorly to therapy after operation and died April 24, 1937.

Autopsy.—Two duodenal diverticula were found, one on each side of the ampulla of Vater. They were approximately the same size, each measuring about 3 cm. in depth by 2 cm. in maximum diameter. The diverticulum behind, and to the right of the ampulla, appeared acutely inflamed and had ruptured at its apex into the retroperitoneal tissue behind the head of the pancreas resulting in extensive multiple retroperitoneal abscesses. The infection involved particularly the tissue along the root of the mesentery and about the right kidney and ureter. The ampulla itself showed no gross abnormality.

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The only other finding of significance was an extensive bilateral, confluent bronchopneumonia.

COMMENT.—Spontaneous rupture of a duodenal diverticulum is apparently a rare occurrence. A search through the English literature on duodenal diverticula revealed a case which was reported in 1926 by Monsarrat[1]. This patient, a white female, age 58, gave a long history of "indigestion." A preoperative diagnosis of acute cholecystitis was made, but at operation a ruptured duodenal diverticulum was found, removed, and the patient recovered.

In 1930, Lucinian[2] also reported a case of a diverticulum of the duodenum which had perforated into the pancreas. The diagnosis was made preoperatively by roentgenologic examination and was confirmed at operation. Recovery, following removal, was uneventful.

Huddy[3] reports a case of gangrenous diverticulitis in a white female, age 27, mistaken for an acutely inflamed appendix lying under the liver. At operation a duodenal diverticulum was found, black and necrotic at the tip. The diverticulum was excised and the patient recovered. There may have been some leakage from the diverticulum in this case but no acute perforation was found.

REFERENCES

[1] Monsarrat, K. W.: Acute Perforation of a Duodenal Diverticulum. British Jour. Surg., **14**, 179, 1926–1927.

[2] Lucinian, Joseph H.: Diverticulum of the Duodenum Perforated into the Pancreas. Am. Jour. Roentgenol. and Radium Ther., **24**, 684, 1930.

[3] Huddy, G. P. B.: Duodenal Diverticula with Report of a Case of Gangrenous Diverticulitis. Lancet, **2**, 327, 1923.

A MODIFICATION OF THE INCISION FOR THORACOPLASTY*

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THE PRESENT-DAY operations for thoracoplastic compression of the tuberculous lung differ materially from those of Sauerbruch,[1] Brauer[2] and others,[3] beginning about 1908. The first important modification for reducing the gravity of the operation was that of Maurer,[4] who did not carry his incision higher than the spine of the scapula and still was able to resect the first rib by upward retraction of the skin and mobilization of the deeper tissues. About three and one-half years ago the writer[5] published a method in which the lower end of the Maurer incision was carried forward in the seventh interspace just below the angle of the scapula. The upper stage of the thoracoplasty was then replaced by the far more valuable apicolysis. Indeed, it has been found that the first rib need not be sacrificed,[6] provided full compression of the diseased upper lung has been secured. If, then, there are no

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tuberculous lesions in the lower part of the lung, this apicolytic procedure may be all that is necessary. When, however, there is disease of the lower lung and surgical compression seems advisable, it has been the custom heretofore to reopen the lower part of the first-stage scar and to continue the paravertebral incision downward and outward (Fig. 1).

A greatly simplified lower-stage operation is herewith presented, which procedure, apparently, causes the least possible trauma.

Since, at the first stage, the seventh rib is within easy reach and the incision has been carried forward around the angle of the scapula, it is unnecessary to reopen this wound. By now incising down upon the ninth or the tenth rib, and retracting the latissimus dorsi and the spinal muscles backward without transversely dividing them, there will be afforded space enough for the completion of the thoracoplasty. Through the incision upon the ninth rib as much of the bone as is necessary may be taken away, just as is usually accomplished in the simple operation for empyema. Then the eighth rib, if still present, may be exposed by retracting upward and the tenth by retracting downward. If it is desired to take the eleventh rib also, the incision may be made upon the tenth instead of the ninth; or still another may be made upon the eleventh rib or in its interspace.

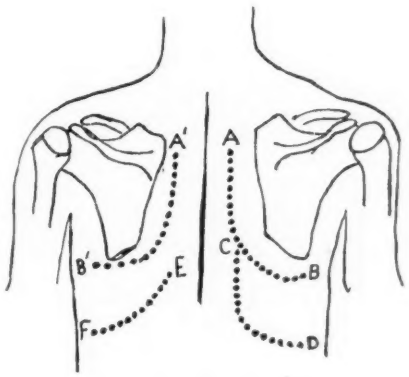

FIG. 1.—A—B and A′—B′: Maurer's incision for upper-stage thoracoplasty or for apicolysis (slightly modified). C—D: Incision for lower thoracoplasty. E—F: Author's incision for lower-stage thoracoplasty.

These rib-parallel incisions with muscle retraction, as here described, are far less traumatizing than those which cross the ribs at any angle.* They can nearly always be accomplished under local anesthesia. The postoperative compression is, of course, managed according to the principles for thoracoplasty.

The well-known rule that two shorter wounds are less shocking and less subject to complications than a single long one, holds good in thoracic as well as in abdominal or other surgical procedures.

REFERENCES

[1] Sauerbruch, F.: Die chirurgische Behandlung der Lungentuberkulose. Verhandl. des VI Internat. Tuberkulosekongresses, 1908.

[2] Brauer, L.: Erfahrungen und Ueberlegungen zur Lungenkollapstherapie. Beitr. z. Klinik. d. Tuberkulose (Lit.-Verz.), **xi**, 1909.

[3] Friedrich, P. L.: Die operative Beeinflussung einseitiger Lungenphthise durch total Brustwandmobilisierung und Lungenentspannung. Arch. f. klin. Chir., 2-38, 1908. Also later works.

* All the usual incisions except that of Maurer are diagrammatically demonstrated in Dr. John Alexander's recent excellent book on collapse therapy[7].

[4] Maurer, A.: Le Thoracoplastie dans le traitement de la tuberculose pulmonaire. J. de Chir., **36**, 857, 1930.

[5] Lilienthal, Howard: Pulmonary Tuberculosis; Recent Types of Operation. J.A.M.A., **102**, 1197, 1934.

[6] Lilienthal, Howard: Conservation of the First Rib in Apicolytic Thoracoplasty. Jour. Thor. Surg., **6**, 414, 1937.

[7] Alexander, John: The Collapse Therapy of Pulmonary Tuberculosis, 473. Charles C. Thomas, Springfield, Ill.

DISCUSSION.—DR. OTTO C. PICKHARDT (New York) said that there is as yet no standard incision for thoracoplasty, in the sense that a McBurney, Kammerer, or suprapubic incision is standardized, and, therefore, any new idea is of course welcome. In general, the thoracic surgeon performing a thoracoplasty makes a long curved incision somewhere between the scapula and the spinous processes from above downward, extending it upward for the higher ribs, and downward and forward for the seventh and eighth ribs, around the lower tip of the scapula. This is usually found in the seventh intercostal space. The name "periscapular incision" has been suggested for this. The muscularity of the patient and the amount and number of ribs to be resected are, of course, a guide to the length of the cut. The higher the ribs to be resected, the more muscles there are which have to be actually cut across. The lower down the ribs are, the more varied and the easier it is to remove them. The names of Brauer, Friedrich, Wilms and Sauerbruch are attached both to their incision and the type of operation. For the eighth, ninth, tenth and eleventh ribs, a hockey-stick type of incision, separate from the first one and placed more medially and lower, is employed. Emile Holman has employed a posterior curved incision added to the lower third of the primary incision. It is in the third and fourth stages, and in the eventual removal of the anterior portions of the unresected ribs that ingenuity must be exercised in order to cause the least possible damage to muscles and nerves.

AN EFFICIENT METHOD FOR THE REDUCTION AND IMMOBILIZATION OF COLLES' FRACTURE*

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TABULATION of the examination of 125 claimants, previously involved in an accident, whose alleged injury was a fracture of the wrist, has shown some interesting facts which have led to changes in our treatment of these fractures. In 102 of these, the examination was made between three and nine months after the accident. More than 75 private physicians, whose names were recorded, and 27 different hospitals rendered the surgical care, so the final results are representative of those now commonly obtained in New York City. The results were good in 82 cases, or two-thirds of the total.

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Twenty-two had fair results and 21 were poor. Wrist flexion was impaired 20 per cent or more in half of all the cases while extension of the wrist was normal in two-thirds and only a small percentage of restriction was present in the remaining third. Pronation of the wrist was seldom diminished, but supination could not be fully obtained in 22 individuals, or nearly one-

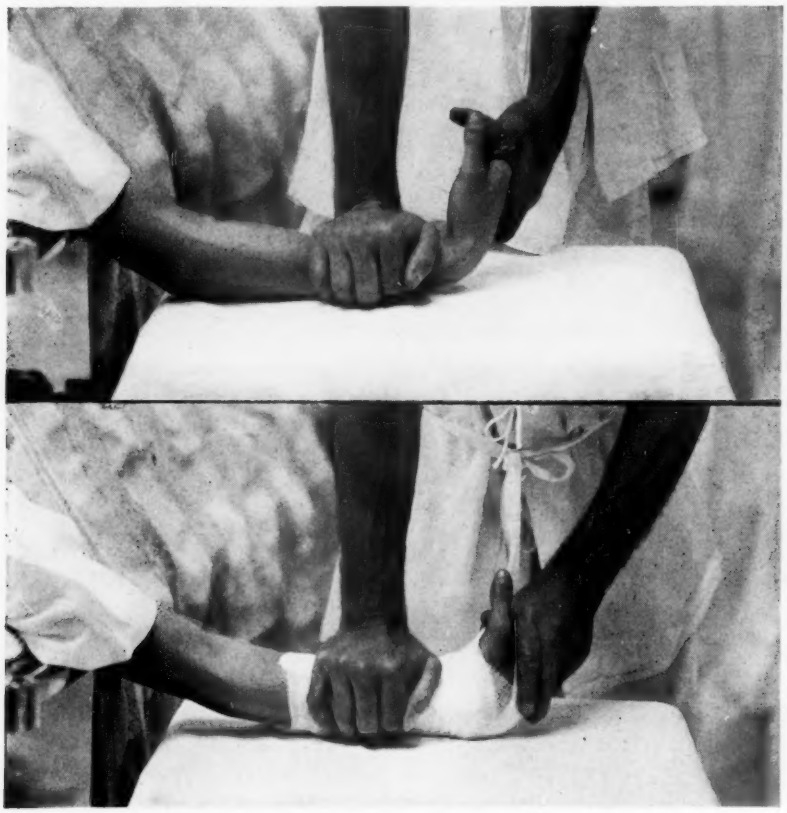

FIG. 1.—Illustrates the method of reduction.
FIG. 2.—Shows the hand being held in position while the plaster hardens.

fifth of the total. Finger flexion was similarly impaired in one-fifth of the cases. Definite deformity, due to anterior displacement of the head of the ulna, was present in 30, and slight deformity in seven others, or nearly one-third of the total. Radial shortening and deviation were found definitely in 27 and slightly in six others, or one-quarter of the total. Failure of a few claimants to prove these fractures in court will merely increase the percentage of unsatisfactory results for the severe injuries need no confirmatory evidence.

To obtain a good result in a Colles' fracture, the first essential is accurate restoration of the displaced bone to its normal position. This is usually attempted by manipulation with the fingers and hands, but is not always successful, for, at times, greater force is necessary.

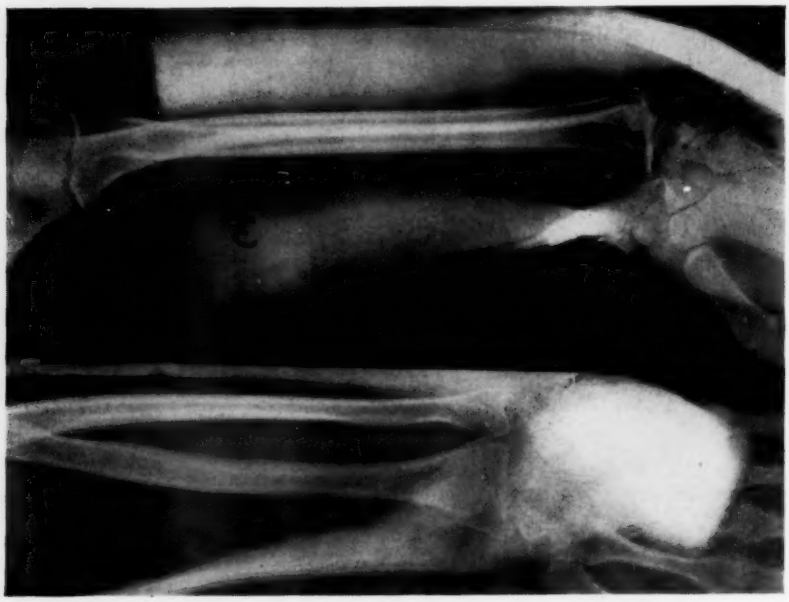

FIG. 3.—Shows overcorrection, the lower fragment of the radius being displaced anteriorly.

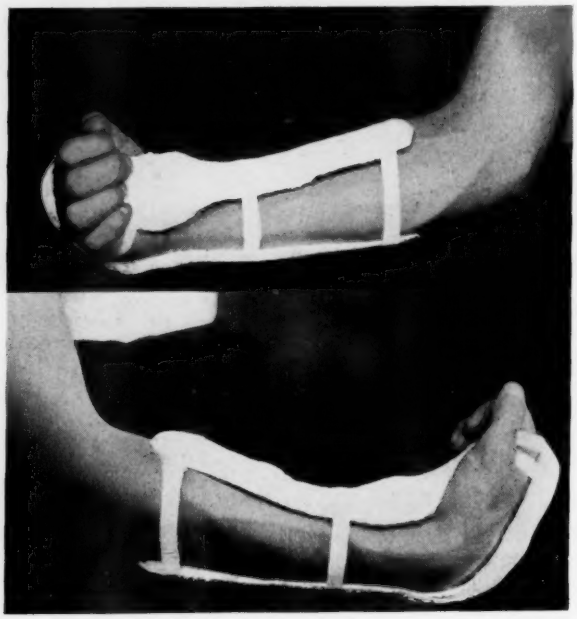

FIG. 4.—Shows wrist flexion, finger flexion and ulnar deviation with rotation of the hand.

FIG. 5.—Shows wrist flexion and pull on the radius with the forearm in supination.

During the past year we have been using the method shown in Figure 1, which substitutes the push and pull of the strong muscles of the shoulder girdle for the weaker ones of the forearm and hand of the operator. This gives better control of the fragments and more perfect and easy reduction of the fracture. Under anesthesia, with the patient in the dorsal position and the injured upper extremity lying upon a table at a right-angle to the body, the surgeon, who is standing in the angle between the body and the arm, places the forearm in complete supination and rests the base of his palm against the lower end of the upper fragment of the broken radius. With his other hand, he then grasps the hand of the patient, flexes the wrist half way and forcibly pulls the lower end of the broken radius downward and forward as he pushes the upper end backward (Fig. 1). A distinct crepitation is usually felt long before the full force available has been used. This indicates a reduction of the dorsally displaced fragment to its proper position. If too much strength is used in this method, an overcorrection or anterior displacement may be produced (Fig. 3). This must be carefully avoided.

After proper alignment has been secured, anterior and posterior plaster splints, narrower than the wrist, but thick enough to be strong, are placed in position and the upper ends held by a few turns of a gauze bandage. With the forearm still in complete supination and the wrist in about two-thirds of normal flexion, the bandage is continued downward over the wrist and hand almost to the base of the heads of the metacarpal bones, where the splints should end. Then, while the plaster is still soft, with the wrist thus flexed and held in supination, the hand is pulled away from the radius toward the ulna, making traction on the radius to maintain its full length as much as possible. The hand is also rotated so as to throw the head of the ulna backward. During these manipulations, the other hand of the operator has held the upper portion of the splints firmly back against the table upon which the extremity rests (Fig. 2). This position is held until the plaster hardens (Figs. 4 and 5). To recapitulate: Complete supination, wrist flexion, hand in ulnar deviation and rotated with the thumb toward the ulna anteriorly.

The fingers and thumb should be flexed and extended from the first day throughout convalescence. The disadvantages of immobilization of the wrist in a flexed position are well known but can easily be prevented by proper care. After one week, the splints should be removed at frequent intervals, the wrist should be firmly grasped by a hand encircling the site of fracture and flexion and extension of the wrist gently performed by the surgeon in ever increasing range as a longer time elapses. The patient should frequently place the forearm in the supine position on a table even while the splints are in place and the sling should be discarded as soon as possible. In a limited number of cases, we have found this an easy method to reduce Colles' fractures and a means of preventing some of the disability and deformity that have followed them in the past. Nothing can take the place of adequate personal attention by the surgeon, and later physical therapy.

The histories of the patients examined show that many of them were kept in splints or in a plaster encasement for a period of six weeks or more with no attempt at regular massage or motion. Undoubtedly, some of the disability and delay in recovery were due to these factors.

An extensive search of the literature has failed to find a description of a similar method for the reduction of Colles' fracture. Buxton[1] says, "Supination is the position of choice in immobilization of fractures of both bones, because it is the optimum position for the recovery of function, as the most important arm movements are from supination to pronation, and after fixation, the latter movement is more easily recovered than supination."

SUMMARY

(1) Examination of 125 individuals at varying intervals after an alleged fracture of the wrist showed impairment of wrist flexion and supination and deformity due to anterior displacement of the head of the ulna and shortening of the radius in a considerable percentage of the cases.

(2) A method previously undescribed, we believe, is proposed for the reduction of Colles' fracture.

(3) A method of immobilization, designed to minimize the disabilities and deformities mentioned above, is described.

(4) The importance of proper care during the period of immobilization is emphasized.

REFERENCE

[1] Buxton, St. J. D.: Proc. Roy. Soc. Med., **50**; Sec. Orthop., **17**, 1926.

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